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CLINICAL STUDIES OF VITAMIN A IN INFANTS AND IN CHILDREN

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During the past three years, a convenient method of estimating vitamin A in small quantities of blood has been developed and applied to the problems of early clinical diagnosis of deficiency of vitamin A and the metabolism of vitamin A in health and in disease.

The clinical diagnosis of vitamin A deficiency, before the development of the characteristic keratomalacia, has been dependent on the recognition of early signs, as described by Blackfan and Wolbach.[1] They pointed out that the most dependable early sign is the appearance of cornified epithelial cells in sites where they do not normally occur, for example, nose, trachea, kidneys and vagina.[2] Yet even this evidence of deficiency of vitamin A may be considered a relatively late sign, as it appears only after weeks or months of almost complete deprivation of vitamin A. Quantitative measurement of night blindness has been proposed as an indirect approach to the early diagnosis of vitamin A deficiency, but present technics require a cooperation which is difficult to obtain from children under 6 years of age. The desirability of a convenient, direct and objective method for the clinical diagnosis of

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1. Blackfan, K. D., and Wolbach, S. B.: J. Pediat. 3:679, 1933.

2. The fundamental effect of deficiency of vitamin A is to cause atrophy of epithelium with substitution of a stratified keratinizing epithelium for the normal epithelial structure. The cornified epithelial cells which serve as the basis for the diagnosis of deficiency of vitamin A are derived from such keratinized epithelium in the sites where it does not normally occur.

vitamin deficiency, also applicable to clinical investigation of the metabolism of vitamin A in health and in disease, is apparent.

Carr and Price [3] have described the formation of a blue color when materials containing vitamin A are treated with a chloroform solution of antimony trichloride. Subsequent experience has indicated a definite association between the blue color and the vitamin A content of a wide variety of materials as determined by biologic assay.[4]

The Carr-Price reaction has gained wide acceptance, although difficulties adhere to its use. The color forms to a maximum almost instantaneously and then immediately begins to fade rapidly, thus making delay in measurement a source of error. Since the concentration of vitamin A in blood is extremely small, little color can be produced by the antimony trichloride reagent from the small amounts of blood obtainable from infants and children. Other substances form blue colors with antimony trichloride, but only the carotenoids are a source of interference in determining the concentration of vitamin A in blood by the Carr-Price reaction. This interference may be eliminated in part by the use of selective color filters, since the maximum absorption by the blue color from the carotenoids is at 590 millimicrons, while that from vitamin A is at 620 millimicrons. The concentration of carotenoids present may be determined colorimetrically because of their yellow color. Then the small amount of blue color contributed by the carotenoids at 620 millimicrons may be calculated, as will be described, and subtracted from the total blue color at 620 millimicrons to give the corrected value for vitamin A.

The intensity of the blue color has been measured by: (1) the Duboscq colorimeter, a copper sulfate solution being used as a standard for comparison; (2) measurement of absorption in a Lovibond tintometer; (3) spectrophotometric analysis. Each of these methods has been found to have disadvantages, such as slowness and complexity in manipulation, the large quantities of original materials required, difficulty in measuring the small amount of rapidly fading color, elimination of interfering colors, errors of subjective origin, expense of apparatus and skill or experience required to use the apparatus accurately.

We have employed a photoelectric colorimeter with selective light filters in the estimation of carotenoids and vitamin A, with these advantages: (1) The rapidly changing blue color may be read instantly at its maximum; (2) subjective error is eliminated; (3) selective light filters allow for correction due to blue color arising from the carotenoids; (4) carotenoids and vitamin A may be determined with the same aliquot of extract; (5) the small amounts of color obtainable from 1 cc. of blood

3. Carr, F. H., and Price, E. A.: Biochem. J. **20**:497, 1926.

4. Morgan, R. S.; Edisbury, J. R., and Morton, R. A.: Biochem. J. **29**:1645, 1935.

serum may be measured accurately; (6) the entire procedure is simple to perform.

METHOD

The method of preparation of extracts of materials for analysis employed is that described by McCoord and Luce-Clausen[5] except that the reagent was made with 22 per cent of antimony trichloride in chloroform. An Evelyn photoelectric colorimeter was used.[6]

PREPARATION OF THE EXTRACT OF BLOOD SERUM (OR PLASMA).[7]—To 1 cc. of serum (or plasma) in a narrow mouth tube,[8] 2 cc. of 95 per cent ethyl alcohol is added and the mixture shaken. Then 2 cc. of purified petroleum benzine U. S. P. (petroleum ether), accurately measured, is added. After the tube is tightly stoppered it must be shaken vigorously and continuously for ten minutes if complete extraction is to be attained. Centrifuging causes the separation of the clear upper layer of purified petroleum benzine extract which is used in the analyses. The layer measures 2 cc., and from it 1 cc. may easily be taken for the analyses.

ANALYSIS OF THE EXTRACTS.—The yellow color of the petroleum benzine extract is due to carotenoid pigments, the bile pigments being unextracted under these conditions. The principal carotenoids in blood serum are carotene and xanthophyll; other carotenoids are present in only meager amounts. Furthermore, the carotene and xanthophyll are usually present in the same proportion, so that measurement of the total carotenoids gives a sufficiently accurate reflection of the amount of carotene present for practical purposes. Estimation of the total amount of carotenoids is necessary in the determination of vitamin A, in order that the measurement of the total blue color formed by the Carr-Price reaction may be corrected for that portion arising from the carotenoids.

CENTER SETTINGS.—Filter 440[9] is placed in the microunit of the photoelectric colorimeter. The center setting of the instrument for 1 cc. of purified petroleum benzine in a 1 cc. open type microabsorption cell is determined in the manner described in the directions for operation of the instrument.[10]

A similar "center setting" is determined for the antimony trichloride reagent with filter 620. It is important to redetermine the center settings immediately before each analysis. One cubic centimeter of the reagent should be accurately pipetted into a 1 cc. open type cell. The pipet used to measure the antimony trichloride reagent should be calibrated to deliver exactly 1 cc. within four

5. McCoord, A. B., and Luce-Clausen, E. M.: J. Nutrition **7**:557, 1934.

6. Evelyn, K. A., and Cipriani, A. J.: J. Biol. Chem. **117**:365, 1937.

7. Vitamin A and carotenoids have not been found in appreciable amounts in the blood corpuscles.

8. Extraction tubes: Loss of the volatile extracting solvent during the extraction procedure would introduce serious error; consequently, precautions to prevent evaporation are essential. Best quality cork stoppers should be used. Tubes measuring 16 by 10 mm. with a flange at the opening 15 mm. long, having internal diameters of 8 mm. at the base and 10 mm. at the top, have proved satisfactory.

9. Filter numbers refer to their maximum transmission, that is, no. 440 at 440 millimicrons and no. 620 at 620 millimicrons.

10. Evelyn, K. A.: J. Biol. Chem. **115**:63, 1936.

seconds by gravity, with its tip steadied against the side of the cell 3 mm. from the top, the last drop being blown out with a gentle puff. This is to insure accuracy and rapidity in the technic and to avoid spilling of the liquid or immersion of the tip of the pipet in the reacting mixture. All operations must be performed quickly, but accurately and smoothly.

CAROTENOIDS.—With filter 440 in place and the instrument set at the appropriate "center setting" for purified petroleum benzine, 1 cc. of the petroleum benzine extract of the serum is accurately measured into a 1 cc. open type cell in place in the cell holder. The cell is immediately wound into the colorimeter and the galvanometer reading (G440) recorded. This reading is used in calculating the total amount of carotenoids present.

VITAMIN A.—The 1 cc. cell with its contents is carefully removed from the colorimeter without spilling any of the extract. The petroleum benzine is now evaporated to dryness by gently blowing dry air into the cell while it is held partially immersed in water kept at from 40 to 45 C. The use of a Petri dish with a filter paper in the bottom is convenient and prevents scratching of the polished bottom of the cell. The outside of the cell is then wiped dry and polished with lens paper and the cell replaced in the cell holder of the colorimeter. Filter 620 is put into the colorimeter, and the galvanometer is set at the "center setting" for the antimony trichloride reagent. When everything is ready, 1 cc. of the antimony trichloride reagent, accurately measured, is pipetted into the cell, and it is immediately wound into the colorimeter, rapidly but smoothly, to avoid spilling of the cell's contents.

The blue color comes to its maximum formation in a few seconds. The galvanometer reading (G620) is taken at the point of maximum absorption of light, that is, maximum color formation.

EXPRESSION OF RESULTS.—Pure vitamin A was not available for calibration of the photoelectric colorimeter to allow the results to be expressed as U. S. P. units or milligrams of vitamin A. There is no advantage in converting the galvanometer readings into international units by means of a factor calculated from calibration data obtained from using some impure concentrate of vitamin A. Therefore, the results are expressed in abstract units directly related to the galvanometer readings and readily convertible into U.S.P. units when a suitable conversion factor is found. Only results obtained in the 1 cc. microcolorimeter cell and following the conditions of extraction described can be compared.

Our results, obtained using the Evelyn colorimeter, are expressed in terms of units calculated as follows:

$$L = 2 - \log. G \text{ (galvanometer reading)}$$

$$\text{Units per cubic centimeter} = L \times \frac{2}{\text{number of cubic centimeters of serum analyzed}}$$

$$\text{Units} \times 100 = \text{units per hundred cubic centimeters.}$$

Results for carotenoids are designated as 440 units and for vitamin A as 620 units, indicating the maxima of the light band by which the color intensity is measured.

CALCULATION OF 620 UNITS OF VITAMIN A.—The value of the 620 units of vitamin A is calculated by subtracting the number of 620 units of blue color contributed by the carotenoids from the total number of 620 units of blue color formed by the antimony trichloride reagent.

Each 440 units of carotenoids produces 0.11 of a 620 unit of blue color with antimony trichloride. This factor was obtained by determining the ratio of $\frac{440 \text{ units}}{620 \text{ units}}$ for mixtures of carotene and xanthophyll in the proportions usually present in blood serum.[11]

Example: 1 cc. of purified petroleum benzine extract from 1 cc. of blood serum was analyzed.

G440 = 71
G620 = 81
L440 = 0.1487
L620 = 0.0915
440 units per cubic centimeter = 0.1487 × 2/1 = 0.2974
440 units per hundred cubic centimeters = 0.2974 × 100 = 29.7 = total carotenoids
620 units per cubic centimeter = 0.0915 × 2/1 = 0.1830
620 units per hundred cubic centimeters = 0.1830 × 100 = 18.3
620 units per hundred cubic centimeters from carotenoids = 29.7 × 0.11 = 3.3
620 units per hundred cubic centimeters from vitamin A = 18.3 — 3.3 = 15.0

TABLE 1.—*Results of Duplicate Analyses of Blood*

Patient	Carotenoids (440 Units)	Difference	Vitamin A (620 Units)	Difference
J. McD.	30.3		16.1	
	28.0	2.7	17.5	1.4
M. C.	25.3		16.7	
	23.3	2.0	17.2	0.5
H. S.	29.7		17.6	
	31.7	2.0	18.5	0.9
A. B.	5.1		5.9	
	5.1	0.0	5.4	0.5
H. H.	11.8		11.1	
	11.4	0.4	10.2	0.9
E. S.	17.7		34.8	
	17.7	0.0	35.1	0.3
B.	22.0		14.9	
	21.6	0.4	15.1	0.2
A.	39.0		9.8	
	39.2	0.2	10.0	0.2
H.	21.5		19.6	
	20.6	0.9	20.0	0.4
B. T.	2.9		5.8	
	2.9	0.0	5.6	0.2
F. H.	22.0		17.0	
	20.9	1.1	18.2	1.2
P. B.	2.8		35.5	
	2.9	0.1	34.9	0.6
Average difference		0.8 unit		0.6 unit
Maximum difference		2.7 units		1.4 units

The accuracy and dependability of this technic are indicated by the results obtained when duplicate analyses for carotenoids and vitamin A were done on separate specimens of the same blood. The average difference in the determination of vitamin A was 0.6 unit and the maximum difference was 1.4 units (table 1), and in the case of the carotenoids the differences were 0.8 and 2.7 units respectively. All variations due to extraction, color formation and color measurement are included in such a comparison.

11. Clausen, S. W., and McCord, A. B.: J. Pediat. **13**:635, 1938.

RESULTS [12]

This technic, permitting the quantitative estimation of vitamin A and carotenoids in the small amounts of blood serum which can be obtained easily from infants and children, has provided a means of exploring the variations of the levels in blood in health and in disease. In these studies emphasis has been placed on the determination of the factors which may result in a deficiency of vitamin A. The usefulness of the levels in the blood in the early clinical diagnosis of deficiency of vitamin A has received special consideration. The indispensable role of vitamin A in maintaining normal growth, along with its other effects, renders the pursuit of exact definition of the conditions affecting its metabolism particularly interesting in infancy and childhood.

We may proceed then to the questions which naturally arise and consideration of the data we have gathered to answer them.

NORMAL LEVELS OF VITAMIN A AND CAROTENOIDS IN THE BLOOD

By means of the technic described, normal well nourished infants and children have been found to have levels of vitamin A in their blood ranging from 5.5 to 27.3 units. A representative collection of values in the blood of normal infants and children from birth to 12 years of age is presented in table 2.

With infants, the almost universal practice of supplementing the vitamin A intake from the diet with vitamin A concentrates makes it difficult to determine the levels in the blood under natural conditions. Nevertheless, we have observed a few infants who had received no additional vitamin A. The levels in these children are shown at the beginning of table 2. The children over 2 years of age had not received any accessory vitamin A since infancy but only a well balanced diet, and yet the levels of vitamin A tend to be higher than in infancy.

It appears that the levels of carotenoids in the blood in normal infants and children also cover a wide range, from 3.1 to 75.7 units. The levels are low before solid foods containing carotenoids are introduced into the diet, and then the levels increase rapidly. Additional evidence of the effects of the intake is supplied by the patient F. H. (table 7), who will be discussed. During a period with concentrates added to the diet this patient had much higher levels than in a subsequent period without added concentrates.

To determine the effect of intake of ordinary amounts of vitamin A and carotenoids on the levels in the blood during absorption, studies were made with infants and children who were ingesting the quantity

12. Carotene may be converted into vitamin A by the body. Therefore, it is important that in the consideration of the significance of the level of vitamin A in the blood the amount of the carotenoids present also receives attention.

TABLE 2.—*Concentrations of Vitamin A and Carotenoids in the Blood of Normal Infants and Children**

Age	Carotenoids (440 Units)	Vitamin A (620 Units)	Age	Carotenoids (440 Units)	Vitamin A (620 Units)
4 days	6.5	10.2	5 yr. 6 mo.	40.5	16.2
3 wk.	6.1	8.9	7 yr.	35.8	18.0
3 wk.	6.1	7.2	7 yr.	59.0	16.2
4 wk.	3.5	5.9	7 yr. 3 mo.	32.8	12.2
6 mo.	6.5	10.9	7 yr. 6 mo.	35.8	15.7
			7 yr. 8 mo.	36.9	13.7
6 wk.	3.1	7.1	8 yr.	38.1	13.3
8 wk.	5.1	5.5	8 yr.	35.1	9.4
2 mo.	5.3	13.0	8 yr.	25.6	19.9
2 mo.	7.5	13.0	8 yr.	29.7	8.1
5 mo.	7.2	8.8	8 yr.	39.5	21.5
9 mo.	34.8	19.3	8 yr.	42.2	11.5
11 mo.	48.8	16.2	8 yr. 6 mo.	44.4	13.4
1 yr.	38.5	13.1	8 yr. 6 mo.	50.4	12.3
1 yr. 6 mo.	51.5	19.9	9 yr.	49.6	12.7
2 yr.	72.4	16.7	9 yr.	32.2	9.6
2 yr. 6 mo.	46.2	9.0	9 yr.	51.9	13.9
2 yr. 6 mo.	72.4	17.6	9 yr.	43.5	11.8
2 yr. 6 mo.	46.2	12.4	9 yr.	66.4	17.1
3 yr.	36.4	10.1	9 yr.	34.8	11.1
3 yr.	72.4	14.7	10 yr.	37.8	13.4
3 yr. 6 mo.	36.8	11.4	10 yr.	42.6	27.3
3 yr. 6 mo.	51.5	19.2	10 yr.	37.4	11.5
3 yr. 9 mo.	75.7	15.7	10 yr.	46.6	17.0
4 yr. 4 mo.	49.8	11.4	10 yr.	32.5	22.8
4 yr. 6 mo.	42.6	18.0	10 yr. 4 mo.	37.0	16.0
5 yr.	36.2	13.9	10 yr. 5 mo.	49.5	13.4
5 yr.	43.6	10.6	10 yr. 6 mo.	56.0	20.9
5 yr.	67.4	14.7	10 yr. 8 mo.	10.5	9.4
5 yr. 6 mo.	28.2	11.0	11 yr. 6 mo.	30.7	16.8
5 yr. 6 mo.	19.4	10.2	12 yr.	30.4	15.3
5 yr. 6 mo.	25.3	21.6	12 yr.	30.0	12.1

* In the first 5 infants no accessory vitamin A had been given. In the remainder of the infants the vitamin A intake from the diet had been supplemented at some time by the addition of a vitamin A concentrate. In no instance was the sample of blood taken within twenty-four hours of the administration of such a concentrate. The children over 2 years old had not received accessory vitamin A since infancy. Carotene concentrates were not received at any time by any of the patients.

TABLE 3.—*Diurnal Variations in the Levels of Vitamin A and Carotenoids in the Blood of Normal Infants and Children Not Receiving Accessory Vitamin A or Carotenoids* *

Patient	Time of Taking Specimen	Carotenoids (440 Units)	Vitamin A (620 Units)
A. C.	9:30 a. m.	29.7	9.4
	12:00 noon	30.4	9.8
	2:30 p. m.	30.1	8.3
	6:00 p. m.	29.7	9.6
	10:00 p. m.	30.4	9.8
Maximum variation		0.7 unit	1.5 units
R. F.	10:00 a. m.	51.5	18.5
	11:00 a. m.	49.6	18.2
	12:30 p. m.	51.1	21.7
	4:00 p. m.	50.9	21.4
	6:00 p. m.	50.4	20.1
Maximum variation		1.9 units	3.5 units
C. M.	12:15 p. m.	60.6	21.5
	6:00 p. m.	58.5	20.3
	10:30 p. m.	56.5	19.0
	8:00 a. m.	57.7	19.4
Maximum variation		4.1 units	2.5 units
C. W.	9:30 a. m.	31.6	27.6
	12:00 noon	32 2	28.4
	2:00 p. m.	29 7	29.7
	6:00 p. m.	28.2	26.6
	10:00 p. m.	27.6	27.1
Maximum variation		4.6 units	3.1 units

* A well balanced diet was being ingested, containing butter, milk, eggs and vegetables but no accessory vitamin A or carotenoids.

of vitamin A and carotenoids contained in a well balanced diet, including the usual amounts of butter, milk, eggs and vegetables. The minor alterations in the levels in samples of blood taken at various times during the day, before and after meals, may be seen in table 3.

TABLE 4.—*Daily Variations in the Levels of Vitamin A and Carotenoids in the Blood of a Normal Person**

Date	Carotenoids (440 Units)	Vitamin A (620 Units)
1/ 2/40	47.3	20.7
	46.6	20.2
1/ 4/40	45.1	18.0
	44.0	19.0
1/ 6/40	47.3	20.4
	48.4	20.9
1/ 8/40	42.9	19.4
	42.6	18.8
1/10/40	43.6	19.9
	44.4	20.4
1/12/40	43.6	21.4
	44.0	20.8
Maximum deviation from mean	3.4 units	2.0 units

* The blood samples were analyzed in duplicate. A well balanced diet was being ingested, containing butter, milk, eggs and vegetables but no accessory vitamin A or carotenoids.

TABLE 5.—*Diurnal Variations in the Levels of Vitamin A and Carotenoids in the Blood of Normal Children Receiving Usual Amounts of Vitamin A Concentrate**

Patient	Time of Taking Specimen	Carotenoids (440 Units)	Vitamin A (620 Units)
R. C.	8:50 a. m.	21.9	7.1
	12:00 noon	21.9	21.8
	2:00 p. m.	21.0	12.4
	4:00 p. m.	23.3	11.3
	6:00 p. m.	22.1	11.5
	8:00 a. m.	22.4	6.5
Maximum deviation		2.3 units	15.3 units
J. R.	8:50 a. m.	14.9	8.0
	12:00 noon	15.7	21.3
	2:00 p. m.	16.2	19.2
	4:00 p. m.	15.9	14.0
	6:00 p. m.	16.4	11.0
	8:00 a. m.	15.9	7.2
Maximum deviation		1.5 units	14.1 units

* These infants each received 10 drops of percomorph liver oil at 9:00 a. m. in addition to a formula of whole milk.

Observations were made of the levels of carotenoids and vitamin A in the blood of a person from day to day, extending over a period of ten days. The minimal changes are illustrated in table 4.

However, when large amounts of vitamin A were ingested, as when cod liver oil or concentrates were given, considerable transitory increases occurred in the level of vitamin A in the blood. Even the amounts customarily given to infants, for example, 10 drops of percomorph liver oil, cause a rise of the levels in their blood of as much as 15.3 units for

a few hours, with a return to the original level in about twenty-four hours. The variations are given in table 5.

It is apparent from the data in table 5 that specimens of blood for analysis must be taken at least twenty-four hours after the administration of vitamin A or carotene concentrates to avoid the alteration of the levels due to absorption.

The wide range of values in normal infants and children is not a source of confusion, as it will be seen later that the alterations in the levels of vitamin A and carotenoids in pathologic states are well beyond the limits seen under normal circumstances. Furthermore, the relative constancy of the levels in a given person enables one to study the fluctuations produced by various factors.

TABLE 6.—*Effect of Acute Infection with Fever on Vitamin A and Carotenoid Content of Blood**

	Day of Observation	Temperature F.	Temperature C.	Carotenoids (440 Units)	Vitamin A (620 Units)
M. W., aged 6 years; lobar pneumonia of 5 days' duration.....	1st†	103	39.4	7.0	1.8
	2d	102	38.8	7.0	1.3
	3d	normal	....	12.6	4.4
	4th	normal	....	16.4	11.5
	6th	normal	....	21.0	15.3
	8th	normal	....	38.1	27.1
A. H., aged 10 years, 5 months, neurosyphilis	1st	98	36.6	49.5	13.4
Malarial therapy begun.....	8th	102	38.8	34.9	6.5
	12th	106	41	18.4	4.3
Antimalarial treatment	17th	98	36.6	24.2	8.6
	24th	98	36.6	40.6	15.3

* No accessory vitamin A or carotenoids were given.
† Day of admission.

THE EFFECT OF INFECTION WITH FEVER ON THE LEVELS OF VITAMIN A AND CAROTENOIDS IN THE BLOOD

In acute infections with fever, the levels of both vitamin A and carotenoids in the blood decrease. The levels may rise spontaneously during the subsidence of the infection to normal values. These changes are illustrated by 1 patient with syphilis of the central nervous system who was treated with malarial fever therapy and by 1 with pneumonia (table 6).

In the presence of an *acute* infection with fever, low levels of vitamin A and carotenoids in the blood should not necessarily be taken as an indication either that there has been an inadequate intake of these substances or that the bodily stores have been depleted.

LEVELS OF VITAMIN A AND THE CAROTENOIDS IN THE BLOOD AS AN AID IN THE DIAGNOSIS OF DEFICIENCY OF VITAMIN A

If the levels of vitamin A and the carotenoids are to serve as an aid to the early clinical diagnosis of deficiency of vitamin A, it is essential to determine the answer to the following questions:

1. Do the levels of vitamin A and carotenoids in the blood become lower than normal when the amounts of these substances in the diet are restricted?

2. Do the levels fall below normal before the earliest clinical signs of deficiency appear?

In considering the effect of intake of vitamin A and carotenoids on the levels in the blood it is necessary to exclude the influence of other factors such as infection or disorders of metabolism. We have studied a group of infants and children who had no infection, fever or other illness at any time but who suffered restriction of their intake of vitamin A and carotenoids because of limitation in the diet, starvation, refusal of food or faulty technic in feeding (table 7).

We learned that when the intake of vitamin A and carotenoids is reduced the levels in the blood decrease. If an abundant supply of vitamin A or carotenoids had been provided in the diet and then extreme restriction of intake occurred, the levels of vitamin A and carotenoids in the blood became lower but did not drop below normal even after a period of weeks. If for any reason the bodily stores of vitamin A and carotenoids are not large, as may be the case in normal young infants, the levels in the blood will fall relatively sooner if the intake is extremely restricted.

The course of events appears to be that as the bodily stores are depleted the carotenoids in the blood decrease, and not until these have become considerably lowered does the level of vitamin A begin to fall. This sequence of events might be expected, since carotene is converted by the body into vitamin A.

In some of the infants whose intake of vitamin A and carotenoids was restricted the levels in the blood dropped considerably below the lowest limits of normal. The low levels in the blood were the only clue to a deficiency of vitamin A and dropped before the appearance of what has heretofore been considered the earliest sign of deficiency of vitamin A, that is, cornified epithelial cells in the conjunctivas and respiratory and genitourinary tracts.

It is of paramount importance to determine the daily intake of vitamin A or carotene required to maintain a normal level of vitamin A in the blood. In this connection the study of F. H. (table 7) is of great interest and suggests that the amount of vitamin A customarily supplied to infants in ordinary whole cow's milk formulas may be scarcely sufficient to maintain an *optimal* level of vitamin A in the blood.

TABLE 7.—*Alterations in the Levels of Vitamin A and Carotenoids in the Blood of Normal Infants with Variations in the Quantities of These Substances in the Diet*

Duration of Observation	Patient	Age	Intake of Vitamin A and Carotenoids	Carotenoids (440 Units)*	Vitamin A (620 Units)
Admission	E. F.	3½ mo.	Much reduced for weeks	....	1.1
7 days			Evaporated milk modification for 1 week; no accessory vitamin A or carotenoids	....	7.3
14 days			Evaporated milk modification; 20 drops of percomorph liver oil daily for 7 days	....	22.2
Admission	R. R.	1 yr. 1 mo.	Much reduced for weeks	....	3.7
5 days			Whole milk formula; 15 drops of carotene in oil† daily for 5 days	13.1	5.1
13 days			30 drops of carotene in oil daily for 8 days	35.7	12.2
16 days			30 drops of carotene in oil daily for 11 days	43.2	11.7
Admission	F. H.	1 mo.	Much reduced since birth	1.7	1.1
8 days			Fat-free milk formula; 20 drops of carotene in oil daily for 3 days	3.0	1.8
16 days			20 drops of carotene in oil daily for 11 days; carotene discontinued; evaporated milk modification begun	47.3	19.2
25 days			5 ounces evaporated milk daily; no accessory vitamin A or concentrates	24.4	19.5
5 wk.			5 ounces evaporated milk daily; no accessory vitamin A or concentrates	12.8	8.0
6 wk.			5 ounces evaporated milk daily; no accessory vitamin A or concentrates	12.6	8.3
2 mo.			5 ounces evaporated milk daily; no accessory vitamin A or concentrates	8.4	8.5
3 mo.			5 ounces evaporated milk daily; no accessory vitamin A or concentrates	7.2	8.6
4 mo.			5 ounces evaporated milk daily; no accessory vitamin A or concentrates	7.2	6.9
Admission	W. D.	7 mo.	Evaporated milk modification; 5 drops of percomorph liver oil daily for 4 mo.	6.8	15.2
11 days			Percomorph liver oil discontined; given fat-free milk for 11 days	3.3	13.8
6 wk.			Percomorph liver oil discontinued; given fat-free milk for 32 days	4.0	13.1
10 wk.			Percomorph liver oil discontinued; given fat-free milk for 59 days	1.8	12.4

* The early analyses could not include estimation of the carotenoids because the proper filter was unavailable. The extracts of the serum were colorless, indicating that only traces of the cartenoids could have been present.

† The carotene in oil contained 0.3 per cent carotene.

These points may be substantiated by more detailed consideration of data we obtained from a group of infants and children who, as has been pointed out, suffered from no disturbance other than malnutrition (table 7).

E. F. (table 7) was brought to the hospital because at 3½ months of age she weighed no more than at birth. The parents had fed the infant irregularly with insufficient food, and no accessory vitamin A or carotenoids had been given. No underlying disease was found, and there were no clinical signs of deficiency of vitamin A. Cornified epithelial cells could not be found in the respiratory tract, vagina or urine. The level of vitamin A in the blood was the low value of 1.1 units. When given an ordinary evaporated milk formula of 60 calories per pound, she immediately began to gain, and the level of vitamin A in the blood rose to 7.3 units. Then 20 drops of percomorph liver oil was added to her food each day, and after seven days the level of vitamin A in the blood rose to 22.2 units. She gained 5 pounds during the next month.

R. R. (table 7) came from an impoverished family. He had never been able to obtain either a normal diet or any accessory vitamin A or carotene. At 13 months of age, he came to the hospital because of a chronic cough and slow gain in weight, having gained only 8 pounds since birth. No disease was found to be present, but in appearance he was malnourished, and although no evidence of vitamin A deficiency could be found, the level of vitamin A in his blood was reduced to 3.7 units. It was decided to improve this low level of vitamin A indirectly by means of carotene. As seen in the table, the level of carotenoids in the blood rose steadily after the carotene was given. The level of vitamin A soon returned to normal and reached a maximum, while the carotenoids continued to rise. He gained weight rapidly, and the cough disappeared. Since the use of carotene to correct a deficit in the body stores of vitamin A depends on its conversion into vitamin A by the liver, improvement may be more gradual than when vitamin A is given. Furthermore, if disease of the liver is present, the effectiveness of carotene will be uncertain.

The patient F. H. (table 7) presented an extraordinary opportunity to study the relation between the levels of vitamin A and carotenoids in the blood and the intake of these substances. He was the first baby, prematurely born, of a young mother, who became baffled by the problem of feeding him. He weighed 4¾ pounds at birth and came to the hospital when 1 month old weighing only 4¼ pounds. He had been fed at the breast, but there seemed to be too little milk; so it was supplemented by a grossly inadequate formula. Feedings were offered every hour and a half. In addition, aerophagia had caused considerable vomiting. No concentrate of vitamin A or carotene had been given. He had severe edema, so that he appeared like a patient with nephrosis. The serum protein was 4.4 Gm. per hundred cubic centimeters. The urine and the concentration of nonprotein nitrogen of the blood were normal. Careful investigation revealed no evidence of any disease except the clinical manifestations of starvation and hypoproteinemia. No clinical evidence of deficiency of vitamin A was apparent. On entry, the vitamin A in the blood was at the low level of 1.1 units. He was given a formula containing fat-free milk so that the only appreciable source of vitamin A would be the concentrates which might be added. Carotene in oil was then added in the relatively large amounts indicated in table 7. The levels of carotene and vitamin A in the blood rose to high values after eleven days. The edema rapidly disappeared, and the level of serum protein became normal when the intake of food was improved. After the bodily stores had been restored, it seemed of interest to know what levels would be maintained if all accessory sources of vitamin A were

removed from the diet and the only intake of vitamin A was from evaporated whole milk. So the infant was then fed a diet composed of evaporated whole milk (4 per cent fat) and sugar, all other sources of vitamin A being excluded.[13] After nineteen days of this diet, the levels of vitamin A and carotenoids fell considerably, to 8.0 and 12.8 units respectively. Then the level of vitamin A became stationary at 8 units, while the carotenoids continued to decrease. Not until after three months, when the carotenoids had dropped to 7.2 units, did the level of vitamin A begin to fall again. During the period of these observations, he enjoyed robust health and had increased to 16 pounds at 5 months of age. Thus, it is apparent that the fall in vitamin A was delayed for three months while the carotenoids were being depleted to help maintain the level of vitamin A in the blood, and finally, as the levels of carotenoids became low, the vitamin A fell in spite of the intake of vitamin A provided by the milk.

The patient W. D. (table 7) was in the hospital because of mental deficiency, but was normal in so far as growth and nutrition were concerned. He had received a normal diet and 5 drops of percomorph liver oil per day for four months. At this time the level of vitamin A in his blood was 15.2 units and that of carotenoids 6.8 units. He was then fed only fat-free milk without added concentrates, thus providing a meager intake of vitamin A. The levels in his blood were followed for ten weeks; at the end of this time the vitamin A was still 12.4 units, a normal value, and the carotenoids had fallen gradually to 1.8 units. This further illustrates the long period at which the level in the blood can be maintained at normal while the intake is being restricted, if the supply of vitamin A has been abundant before deprivation occurs.

ABSORPTION OF VITAMIN A FROM THE INTESTINE

Attention thus far has been focused on the effects of variations in the intake of carotenoids and vitamin A on the levels of these substances in the blood of normal children. If the absorption of vitamin A from the intestine is impaired, depletion of the bodily stores and lowering of the level in the blood may be expected, even when the vitamin A intake is normal.

The study of intestinal absorption has been facilitated by the utilization of a vitamin A absorption test.[14]

A satisfactory procedure is to give a standardized test dose by mouth (0.1 cc. of percomorph liver oil per pound of body weight [14a]), collecting a fasting sample of blood before the test dose and then samples three, five, seven, nine and twelve hours afterward.

When this standardized dose was given to a group of normal infants and children, it was found that the level of vitamin A in the blood would rise 52 to 231 units above the fasting level in from three to five hours; 130 units was the average rise.

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13. The intake of vitamin A provided by this diet may be estimated as 600 U.S.P. units per day at first, increasing to 1,800 U.S.P. units as more milk was consumed when he became older.

14. Chesney, J., and McCoord, A. B.: Proc. Soc. Exper. Biol. & Med. **31**:887, 1934.

14a. Percomorph liver oil contains approximately 60,000 U. S. P. units of vitamin A per gram.

TABLE 8.—*Vitamin A Absorption Tests with Normal Infants and Children*

Age	Vitamin A Levels in the Blood (620 Units)						
	Fasting	After Test Dose of Percomorph Liver Oil					Maximum Rise
		3 Hr.	5 Hr.	7 Hr.	9 Hr.	12 Hr.	
1 mo.....	6.1	58.8	56.6	29.0	....	....	52.7
2 mo.....	31.3	38.2	180.8	76.8	56.8	35.5	149.5
4 mo.....	29.7	113.7	59.4	39.9	30.3	....	84.0
6 mo.....	19.6	153.6	159.2	148.6	77.8	63.6	139.6
1 yr. 3 mo....	14.4	166.9	133.7	127.7	71.7	....	152.5
1 yr. 9 mo....	14.4	129.2	151.9	107.3	72.6	51.7	137.5
2 yr.....	13.3	117.3	109.9	87.5	53.3	49.0	104.0
3 yr. 4 mo....	17.9	64.6	82.3	47.3	41.5	32.0	64.4
3 yr. 9 mo....	22.9	33.5	151.4	224.4	140.2	84.8	201.5
4 yr. 3 mo....	14.9	18.7	37.7	90.9	71.3	40.4	76.0
5 yr. 1 mo....	12.4	144.7	125.8	65.0	48.5	41.7	132.3
7 yr. 2 mo....	12.7	105.4	77.8	76.7	46.2	36.6	92.7
7 yr. 8 mo....	16.6	41.2	138.2	135.0	124.8	....	121.6
7 yr. 11 mo...	13.8	42.7	100.0	244.8	85.1	66.2	231.0
9 yr. 1 mo....	22.2	52.3	220.5	145.9	48.2	31.6	198.3
10 yr. 1 mo....	18.5	111.1	156.1	103.1	86.5	87.1	137.6

Average rise, 129.7 units.

TABLE 9.—*Results of Vitamin A Absorption Tests with Patients with Impaired Intestinal Absorption*

Name	Age	Diagnosis	Vitamin A Levels in the Blood (620 Units)						
			Fasting	After Test Dose of Percomorph Liver Oil					Maximum Rise
				3 Hr.	5 Hr.	7 Hr.	9 Hr.	12 Hr.	
P. K.	5 mo.....	Cretinism	27.4	36.1	31.2	....	28.4	25.6	8.7
J. A.	10 mo.....	Pancreatic fibrosis	0.0	1.1	1.8	2.2	2.2	1.5	2.2
P. D.	1 yr. 6 mo...	Pancreatic fibrosis	6.5	7.7	6.2	8.7	8.7	7.7	2.2
M. C.	1 yr. 10 mo...	Pancreatic fibrosis	14.8	14.8	....	15.6	....	13.9	0.8
M. R.	1 yr.....	Celiac disease	6.4	32.9	....	27.3	18.7	16.4	26.5
M. P.	1 yr. 1 mo...	Celiac disease	4.9	7.2	12.6	8.9	8.5	6.8	7.7
W. M.	1 yr. 9 mo...	Celiac disease	10.3	22.0	19.9	16.0	15.0	14.0	11.7
G. E.	2 yr. 2 mo...	Celiac disease	8.7	8.7	8.0	9.4	10.6	10.4	1.9
E. T.	2 yr. 8 mo...	Celiac disease	8.5	9.9	10.4	....	8.9	7.1	1.9
W. S.	2 yr. 10 mo...	Celiac disease	10.6	15.9	18.3	20.9	....	14.6	10.3
H. K.	3 yr. 1 mo...	Celiac disease	9.6	....	13.1	13.2	12.7	10.2	3.6
J. M.	3 yr. 8 mo...	Celiac disease	7.3	....	13.6	11.9	11.0	11.0	6.3
A. H.	4 yr. 9 mo...	Celiac disease	8.9	9.1	14.2	15.1	12.7	9.9	6.2
G. F.	5 yr. 5 mo...	Celiac disease	13.0	10.1	24.4	18.2	....	16.6	11.4
R. B.	8 yr. 1 mo...	Celiac disease	9.7	....	27.4	19.5	15.1	12.5	17.7
W. C.	8 yr. 6 mo...	Celiac disease	6.1	....	8.9	14.4	17.9	10.0	11.8
				Average rise (units)*.....					8.2

* Average rise in normal absorption tests (table 8) was 129.7 units.

CIRCUMSTANCES UNDER WHICH THE ABSORPTION OF VITAMIN A FROM THE INTESTINE IS IMPAIRED

Since vitamin A is a fat-soluble substance, it might be anticipated that its absorption would be affected by those conditions in which the absorption of fat is disturbed.

Failure of the level of vitamin A in the blood to rise to a normal height after ingestion of the dose of vitamin A used in the absorption test described may be assumed to indicate a decrease in absorption from the intestine, as no instance of abnormally rapid removal of vitamin A from the blood stream has been demonstrated.

Impaired intestinal absorption, as revealed by low absorption curves, has been found in congenital obliteration of the bile ducts, fibrosis of the pancreas,[15] celiac disease and cretinism (table 9).

EFFECTS OF IMPAIRED INTESTINAL ABSORPTION ON THE LEVELS OF VITAMIN A AND CAROTENOIDS IN THE BLOOD

If the intake of vitamin A and carotenoids is no greater than that provided in a normal diet, chronic impairment of intestinal absorption will result in extreme lowering of the levels in the blood, and symptoms of deficiency of vitamin A will eventually appear. J. A. (table 10), the patient with impaired intestinal absorption, had been ingesting a normal diet but had received no accessory vitamin A or carotenoids. When first seen, at the age of 10 months, he had no vitamin A or carotenoids in the blood and had already shown clinical evidence of deficiency in vitamin A, as revealed by large numbers of cornified epithelial cells in the respiratory tract and urine. P. W. (table 10) is another who received no accessory vitamin A or carotenoids and in whom there was considerable lowering of vitamin A in the blood without other detectable evidences of deficiency of vitamin A. Even if the doses of vitamin A customarily added to the diet are provided, impaired intestinal absorption may still prevent the maintenance of normal levels in the blood (patients N. D., D. R., M. P., in table 10). These low levels may be restored to and maintained at normal by the addition of larger quantities of vitamin A to the diet even though the absorption remains impaired (patients J. A., P. W., N. D., P. D., M. P., W. M., M. C., in table 10).

It is important to realize that a low curve in the vitamin A absorption test does not indicate a complete failure of absorption of vitamin A from the intestines but rather indicates a reduction in the efficiency of the process. There is no advantage in parenteral administration of the vitamin, since the defect in absorption may be easily overcome by the *oral* administration of unusually large amounts of vitamin A, preferably

15. Blackfan, K. D., and May, C. D.: J. Pediat. **13**:627, 1938.

in divided doses. In fact, the presence of a low absorption curve must be taken as an indication for the administration of large amounts of vitamin A [16] if normal levels in the blood are to be maintained and deficiency of vitamin A prevented.

Let us give a more complete consideration of our studies with the patient J. A. to illustrate additional important points. This patient

TABLE 10.—*Data for Patients with Diminished Intestinal Absorption of Vitamin A*

Patient, Age	Diagnosis	Duration of Observation	Intake of Vitamin A	Vitamin A Absorption Test Maximum Rise	Levels in Blood	
					Carotenoids (440 Units)	Vitamin A (620 Units)
J. A. 10 mo.	Fibrosis of pancreas	Admission	No accessory vitamin A or carotene	2.2	0	0
		10 days	15 drops of percomorph liver oil* 4 times daily	0.6	0	5.1
P. W. 6 mo.	Congenital atresia of bile ducts	Admission	No accessory vitamin A or carotene	0.2	6.8	2.9
		9 days	15 drops of percomorph liver oil daily	...	7.2	19.1
N. D. 1 yr. 4 mo.	Fibrosis of pancreas	Admission	10 drops of halibut liver oil daily	1.2	3.5	2.5
		1 mo.	10 drops of percomorph liver oil daily	2.8	2.0	6.8
D. R. 3 mo.	Congenital atresia of bile ducts	Admission	1 tsp. of cod liver oil daily for 1 mo.	0	0	1.8
		19 days	No accessory vitamin A or carotene	...	0	0.0
P. D. 1 yr. 1 mo.	Fibrosis of pancreas	Admission	Vitamin A concentrates corresponding to 20 drops of percomorph liver oil daily	1.1	7.7	8.7
		77 days	10 drops percomorph liver oil twice daily	...	2.6	5.9
		95 days	10 drops percomorph liver oil 3 times daily	...	0.5	12.1
M. P. 1 yr. 1 mo.	Celiac disease	Admission	1 tsp. cod liver oil twice daily	6.8	2.0	4.9
		19 days	5 drops percomorph liver oil 3 times daily	...	2.0	11.4
W. M. 1 yr. 9 mo.	Celiac disease	Admission	Cod liver oil	11.7	6.3	10.3
		13 days	No added vitamin	...	2.0	7.5
		26 days	10 drops percomorph liver oil twice daily	9.1	0.4	12.1
M. C. 1 yr. 10 mo.	Fibrosis of pancreas	Admission	1 tsp. cod liver oil 3 times daily for 16 mo.	0.8	1.1	8.8
		54 days	20 drops percomorph liver oil daily	...	1.9	14.8

* Percomorph liver oil contains approximately 60,000 U. S. P. units of vitamin A per gram.

entered the hospital at the age of 10 months because of diarrhea which had existed since birth. Although well developed he was markedly malnourished, having almost no subcutaneous fat. In addition, he was suffering from chronic pneumonia. His history and physical findings led us to suspect that he was similar to the group of patients who have been found to have lost all normal pancreatic tissue through dilation of the acini with inspissated secretion and subsequent fibrosis.[15] Because of his diarrhea, he had never been given cod liver oil or carotene con-

16. 50,000 U.S.P. units of vitamin A per day.

centrates. On entry, there was no vitamin A or carotenoids demonstrable in his blood. The only clinical evidence of deficiency of vitamin A was the finding of numerous cornified epithelial cells in the respiratory tract and urine. A vitamin A absorption test at this time showed practically no rise, indicating a severe impairment of intestinal absorption. The patient ultimately died of bronchopneumonia and the clinical diagnosis of pancreatic fibrosis was confirmed at necropsy.

This patient provided an opportunity to make a systematic study of the interrelations between the level of vitamin A in the blood, the bodily stores and the evidences of deficiency of vitamin A. Particular attention may be directed to several points relative to the metabolism of vitamin A under these circumstances (figure):

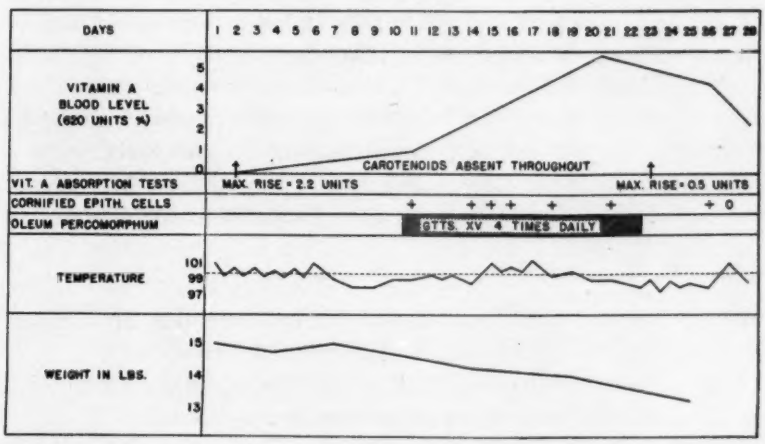

Chart giving data for patient J. A., fully discussed in the text, illustrating the relation between the level of vitamin A in the blood and the evidence of deficiency of vitamin A.

1. When impairment of absorption of vitamin A exists, the amount of vitamin A and carotenoids in an ordinary diet is insufficient to prevent deficiency.

2. The level of vitamin A in the blood reflects this deficiency.

3. Even the earliest clinical sign of established deficiency of vitamin A, cornified epithelial cells, does not appear until the bodily stores are completely exhausted, when vitamin A is absent from the blood.

4. The cornified epithelial cells disappear as the level of vitamin A in the blood reaches the normal range. This indicates cure of the deficiency, as is shown by the fact that no evidence of vitamin A deficiency was found in patient J. A. in microscopic examination of the tissues after necropsy.

5. The repair of the effects of deficiency is relatively rapid. Only ten days were required to restore the level in the blood of patient J. A. to the normal range and to cause the disappearance of the cornified epithelial cells.

6. The carotenoids remained absent from the blood of patient J. A. since too small an amount of these substances was supplied in the diet to be effective under the circumstances of impaired absorption.

7. A large intake of vitamin A must be continued so long as impairment of absorption persists if normal levels in the blood and normal stores in the tissues are to be maintained.

The level of vitamin A in the blood may be maintained at normal by the vitamin A's being consumed before any considerable storage in the body occurs. This is illustrated by the fact that the level in the blood of patient J. A. was soon restored to normal, but as soon as the accessory supply of vitamin A was removed from the diet the level fell rapidly, and shortly afterward, at autopsy, analysis of the liver revealed only meager stores of vitamin A. While the stores of vitamin A in the liver and other tissues may not be essential to maintain a normal level in the blood, they should be looked on as a sort of surplus to be kept adequate to meet any failure of the supply from the diet or increase in the demands for vitamin A.

CONCLUSIONS

A convenient reliable method for the determination of vitamin A and carotenoids in small amounts of blood is described.

This technic has been applied to the clinical investigation of the metabolism of vitamin A in health and disease.

The level of vitamin A in the blood appears to be a useful, direct clinical test for early diagnosis of deficiency of vitamin A. In infants and children deficiency of vitamin A may be detected by a low level of vitamin A in the blood before other reliable clinical signs appear.

The daily intake of vitamin A required to maintain an optimal level of vitamin A in the blood was studied.

Circumstances which increase the requirement for intake of vitamin A are demonstrated.

RESPIRATORY METABOLISM IN INFANCY AND IN CHILDHOOD

XXIII. DAILY ENERGY REQUIREMENTS OF PREMATURE INFANTS

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In a recent paper on infant feeding [1] the statement is made that one of the problems for further study concerns the best method of feeding the premature infant. The lack of unanimity concerning one aspect of the problem, the proper caloric intake, is indicated in two reviews,[2] which state that various authors recommend from 75 to 200 calories or more per kilogram of body weight per day. This wide variability derives from: (1) the difficulty of clinical assessment because of the weak sucking reflex and inadequate manifestations of hunger; (2) the paucity of quantitative data on the total energy exchange of premature infants,[3] and (3) the participation of factors other than diet, such as nursing, environmental conditions and infections, in determining the progress of the premature infant. As part of a systematic investigation of the metabolism of premature infants which may ultimately help to define more clearly their food requirements, the study to be reported here provides quantitative data on the daily energy exchange of 11 premature infants from whom collections were made for a total of one hundred and thirty days and includes analyses of diet, respiratory exchange and excreta.

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Assistance in this work was given by the Children's Bureau of the U. S. Department of Labor.

1. Powers, G. F.: Infant Feeding—Historical Background and Modern Practice, J. A. M. A. **105**:753 (Sept. 7) 1935.

2. (*a*) Tanz, J., and Unger, H.: Beitrag zur Frage des Nahrungsbedarfes der Frühgeburten, Ztschr. f. Kinderh. **59**:135, 1937. (*b*) Peiper, A.: Unreife und Lebenschwäche, Leipzig, Georg Thieme, 1937.

3. Rubner, M., and Langstein, L.: Energie und Stoffwechsel zweier frühgeborener Säuglinge, Arch. f. d. ges. Physiol. **39**:39, 1915.

EXPERIMENTAL PROCEDURE

Subjects.—Eight white and 3 Negro male premature infants in good health were studied in 11 observation periods of from six to twenty-four days each. The birth weight, estimated degree of prematurity, initial nutritional status and average daily diet are given in table 1.

The degree of prematurity as estimated from the expected date of birth varied from zero to fourteen weeks and the weight at birth from 1,130 to 2,220 Gm.

The infants ranged from 10 to 44 days in age and from 1,543 to 2,564 Gm. in weight at the onset of observations. They resided in a room with constant temperature and humidity, with temperatures ranging from 72 to 77 F. and relative humidities from 40 to 80 per cent in different periods of the observations. No difficulty was encountered in maintaining body temperature. Data on water and electrolyte exchange of some of these infants have been reported.[5]

Diet.—The infants were fed mixtures either of human milk or of cow's milk. The former consisted of pooled mother's milk, fresh or frozen, boiled for twenty minutes prior to use. No raw human milk was used. The mixtures of cow's milk consisted of evaporated milk or a powdered skimmed milk–olive oil preparation (Olac) diluted with water and in some observations fortified with dextrimaltose. For 1 infant (R. S.) the preparation contained half of the olive oil usually present in Olac.[6] Each infant received 20 drops of a concentrate either of vitamin D or of vitamins A and D and 10 to 20 mg. of ascorbic acid. The average daily weight gains ranged from 31 to 47 Gm.

Pooled daily aliquot samples of the liquid cow's milk and human milk were analyzed in triplicate for nitrogen (macro-Kjeldahl method), fat[7] and lactose.[8] The powdered preparations were analyzed for nitrogen and fat, the carbohydrate content submitted by the manufacturer being accepted.

The daily caloric intake was determined by multiplying the ingested protein, fat and carbohydrate by the customary constants, 4.1, 9.3 and 4.1.

METHODS

Intake.—The technic of measuring the intake was essentially that devised by Levine for use in the study of water exchange.[9] The powdered ingredients were weighed to 0.1 Gm. by one person and mixed in a small pan with approximately half a measured volume of distilled water. The mixture was transferred to a

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4. Footnote deleted.

5. Gordon, H. H.; Levine, S. Z.; Marples, E.; McNamara, H., and Benjamin, H. R.: Respiratory Metabolism in Infancy and in Childhood: XXII. Water Exchange of Premature Infants—Comparison of Metabolic (Organic) and Electrolyte (Inorganic) Methods of Measurement, J. Clin. Investigation **18**:187, 1939.

6. These preparations were supplied by the Mead Johnson Company.

7. Roese-Gottlieb Method, in Official and Tentative Methods of Analysis Compiled by the Committee on Editing Methods of Analysis, Washington, D. C., Association of Official Agricultural Chemists, 1930, p. 217.

8. Lactose—Official Gravimetric Method of the Association of Agricultural Chemists, in Official and Tentative Methods of Analysis, Compiled by the Committee on Editing Methods of Analysis, Washington, D. C., Association of Official Agricultural Chemists, 1930, p. 216.

9. Levine, S. Z.; Wheatley, M. A.; McEachern, T. H.; Gordon, H. H., and Marples, E.: Respiratory Metabolism in Infancy and in Childhood: XXI. Daily Water Exchange of Normal Infants, Am. J. Dis. Child. **56**:83 (July) 1938.

TABLE 1.—*Clinical Criteria of Prematurity,* Nutritional Status at Beginning of Experimental Period and Average Daily Diet of Infants*

Subject	Weight at Birth, Gm.	Estimated Degree of Prematurity,† Weeks	Period of Observation‡	Number of Periods	Number of Days	Nutritional Status				Average Daily Diet				
						Initial Age, Days	Initial Weight, Gm.	Initial Height, Cm.	Average Daily Weight Gain, Gm.	Fluid, Gm. per Kg.	Calories per Kg.	Calories (%) Derived from		
												Protein	Fat	Carbohydrate
			1937											
N. O.	1752	..	3/15-3/30	4	12	17	1912	44.5	31	161	114	12¶	47	41
E. M.§	1950	5	4/4-4/15	2	11	25	2083	46.0	32	143	113	16¶	44	40
L. O.‖§	1960	..	11/2-11/13	4	9	31	2266	46.0	47	184	123	14	29	57
			1938											
H. L.§	2170	0	1/28-2/12	2	10	23	2564	47.0	27	141	135	18	37	45
E. C.	1920	0	3/17-4/1	2	11	13	2048	45.5	35	142	126	16¶	41	43
R. L.	1200	14	4/7-4/30	4	20	41	1578	41.5	39	134	118	18¶	38	44
M. B.	1500	6	5/12-6/10	3	24	18	1716	42.0	31	213	144	6#	51	43
A. P.	2220	5	10/13-10/24	2	9	17	2379	47.5	34	135	104	20	35	45
R. E.	2030	1	11/8-11/19	3	9	10	2027	46.0	41	155	116	20	35	45
G. C.	1130	12	12/6-12/24	4	9	44	1543	41.0	39	152	118	18¶	37	45
			1939											
R. S.	1900	6	4/13-4/24	3	6	14	2066	44.5	46	164	107	20	20	60

* Roentgenograms of the lower extremities were obtained for all infants except N. O. and E. M. either just before (E. C., H. L., M. B., R. E., G. C., R. S.) or just after study (L. O., R. L., A. P.). The distal epiphysis of the femur was present in all infants except E. C. The proximal epiphysis of the tibia was absent in all except H. L. and R. L. A roentgenogram of the foot was available only for infants H. L., R. L., M. B., R. E., G. C. and R. S. The cuboid bone was absent in all except G. C., in whom it was barely visible. The distal epiphysis of the femur usually appears after the thirty-fifth to fortieth week of gestation, the proximal epiphysis of the tibia and the cuboid bone at the fortieth week (Growth and Development of the Child: I. General Considerations, White House Conference on Child Health and Protection, New York, Century Company, 1932, sect. 1, p. 67).

† The degree of prematurity was estimated on the basis of the mother's history.

‡ With one exception, E. M., the infants were not studied continuously throughout the period of observation. For example, N. O. was studied for twelve days during the period from 3/15/37 to 3/30/37.

§ Negro.

‖ Twin.

¶ Human milk was used in one period of each of these observations.

Human milk was used throughout this observation.

pitcher, the pan and mixing spoon being rinsed with the remainder of the water. The pitcher was then weighed, quickly emptied into six or eight feeding bottles and reweighed. The pitcher, pan and spoon were rinsed into a Kjeldahl flask and the nitrogen content of the washings determined. From the total weight of the formula leaving the pitcher, the original weight of the powdered cow's milk preparation and the nitrogen washings, the total weight of water and solids in the "formula as originally prepared" could be calculated. Individual bottles with rubber caps were weighed before and after each feeding, and the weight of the formula adhering to the sides of the bottles was thus determined. Prior to the day's observation a rack containing the bottles with formula, two sealed empty jars, a package of feeding bibs and a package of gauze were all weighed together. During the feeding of an infant, his lips and the inside of nipples and rubber caps were wiped with sterile gauze, which was immediately placed in the sealed jars. Formula regurgitated on the bibs was similarly handled. At the end of the twenty-four hour period the rack containing the now empty bottles, the two sealed jars containing used gauze and bibs and the remaining unused gauze and bibs were all weighed together. The difference between original and final weights was accepted as the weight of "formula ingested and retained."

Assuming that the formula was homogeneous, one could from the ratio of the weight of "formula ingested and retained" to the weight of "formula as originally prepared" and from the composition of the latter determine the intake of each ingredient. In these observations the error in intake, if one had accepted the "formula as prepared" as the true intake, would have averaged 5 per cent[10] of the true intake and would have ranged from 3 to 10 per cent; if estimated from the weight of the formula leaving the bottles, the average error would have

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10. Assessment of the magnitude of error permits correction of previously reported figures for nitrogen retention in premature infants (Gordon, H. H.; Levine, S. Z.; Wheatley, M. A., and Marples, E.: Respiratory Metabolism in Infancy and in Childhood: XX. The Nitrogen Metabolism in Premature Infants—Comparative Study of Human Milk and Cow's Milk, Am. J. Dis. Child. **54**:1030 [Nov.] 1937). This correction does not alter the conclusions previously reached, which indicate that human and cow's milk dietaries of the same protein and caloric intake permit similar nitrogen retentions. The corrected retentions are 0.23 and 0.26 Gm. of nitrogen per kilogram of body weight per twenty-four hours, respectively, instead of the previously reported 0.25 and 0.27 Gm. The increased nitrogen retention due to doubled and quadrupled intake was decreased from 0.35 to 0.32 Gm. and from 0.45 to 0.39 Gm. per kilogram by the correction. The combined results of previously reported and unpublished data for a total of one hundred and seventy-nine days show a mean nitrogen retention of 0.229 ± 0.006 Gm. per kilogram per twenty-four hours for 14 observations in which protein comprised 6 to 10 per cent of the calories ingested, and a mean of 0.315 ± 0.009 Gm. per kilogram per twenty-four hours for 14 observations in which protein comprised 13 to 20 per cent of the calories. The increase of 0.086 Gm. is almost eight times the probable error of the difference. Paffrath and Bauer (Ueber das Verhalten das Chlor- und Wasserstoffwechsels von drei frühgeborenen und einem ausgetragenen Säugling beim Salzstoss, Ztschr. f. Kinderh. **58**:411, 1936) have suggested subtraction of 5 per cent from the intake in calculating retentions, but the range of 3 to 10 per cent just pointed out makes it obviously more desirable to determine actual intake whenever possible, particularly in comparing the retentions of substances at different planes of intake.

been 4 per cent. The average regurgitation was then only 1 per cent of the true intake, but it should be stressed that the nurses had no other responsibilities than the care of the single infant under observation.

Urine and Feces.—Urine and feces were collected separately and analyzed for nitrogen and for nitrogen and fat, respectively. Urinary nitrogen was used for determination of the nonprotein respiratory quotient. Fecal fat and nitogen were multiplied by the customary constants 9.3 and 25.63 to determine fecal calories. No excreta were examined in the bomb calorimeter.

Respiratory Metabolism.—The daily caloric expenditure was determined for 9 infants [13] by combining the results of twenty-eight calorimetric observations of sixty-eight to three hundred and fifty-five minutes each with the twenty-four hour, minute to minute activity records. In a previous study of 3 full term infants [15] the respiratory exchange was determined in eight observation periods of twenty to twenty-two hours each; in the present study the respiratory exchange of individual infants was determined during their sleep and activity for periods totaling from four to thirteen hours, and on the basis of daily activity records the heat production was calculated for twenty-four hours. Although this method is open to theoretic objection, its use permitted study of a larger number of infants over a longer period and under a more natural environment than the confines of a respiratory chamber. Factors contributing to the choice of method have recently been discussed,[16] and the general reliability of this method has been confirmed by the close agreement between water balances calculated by "metabolic" and "electrolyte" methods.[5]

The closed circuit apparatus used for calorimetric observations on infants L. O. and H. L. consisted of a small chamber for premature infants attached to a Benedict universal respiration table.[17] For all subsequent observations an open

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11 and 12. Footnotes deleted.

13. For infants N. O. and E. M. the total calories were predicted from the insensible perspiration on the assumption that 25 per cent of the heat produced by premature infants is lost by vaporization of water (Gordon, H. H., and Kelly, M. D.: Respiratory Metabolism in Infancy and in Childhood: XIX. The Respiratory Exchange in Premature Infants, Elimination of Water Through the Skin and the Respiratory Passages, Am. J. Dis. Child. **52**:1100 (Nov.) 1936. Gordon and others [5]).

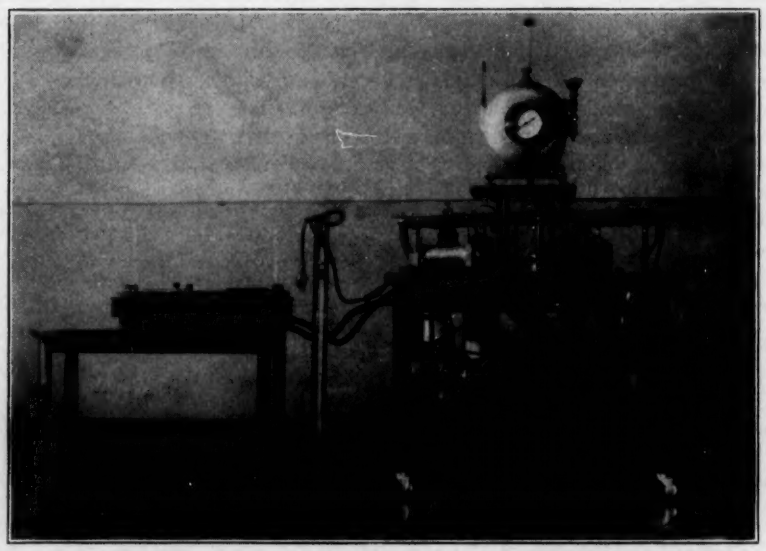
14. Footnote deleted.

15. Levine, S. Z.; McEachern, T. H.; Wheatley, M. A.; Marples, E., and Kelly, M. D.: Respiratory Metabolism in Infancy and in Childhood: XV. Daily Energy Requirements of Normal Infants, Am. J. Dis. Child. **50**:596 (Sept.) 1935.

16. (*a*) Mitchell, H. H.: Some Problems in the Study of Energy Metabolism, in Report of the Conference on Energy Metabolism Under the Auspices of the Committee on Animal Nutrition, Washington, D. C., National Research Council, 1935, p. 66. (*b*) Levine, S. Z., and Wheatley, M. A.: Respiratory Metabolism in Infancy and in Childhood: XVII. The Daily Heat Production of Infants; Predictions Based on the Insensible Loss of Weight Compared with Direct Measurements, Am. J. Dis. Child. **51**:1300 (June) 1936.

17. Gordon, H. H., and Levine, S. Z.: Respiratory Metabolism in Infancy and in Childhood: XVIII. The Respiratory Exchange in Premature Infants; Basal Metabolism, Am. J. Dis. Child. **52**:810 (Oct.) 1936.

circuit apparatus [18] resembling the systems described by Higgins [19] and Newburgh and his co-workers [20] was used. This apparatus consists of the same small chamber attached to a table with devices for circulating and sampling the air in the system. By means of a blower, air is drawn from a ventilator through five bottles containing calcium chloride, soda lime and calcium sulfate. The preliminary removal of carbon dioxide permits use of ventilator air in the air-conditioned room where the apparatus is located, and the removal of water permits subsequent determination of water vaporized by the infants. The air is drawn through the chamber and an aliquot sample removed by the master pump described by Barker.[21] Analyses of the incoming air were made by the Carpenter-Haldane technic [22] before each observation. The average oxygen content for the incoming air in 193 samples was 20.935 per cent, with a standard deviation of 0.006. The average carbon dioxide content was 0, with a standard



Open circuit apparatus used in measuring the respiratory metabolism of the premature infants under observation.

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18. This apparatus was constructed by Mr. R. E. Phipps and Mr. E. Smyth after consultation with Dr. S. B. Barker and Dr. W. H. Chambers, of the department of physiology of Cornell University Medical College.

19. Higgins, H. L., and Bates, V.: New Method for the Determination of the Basal Metabolism of Babies and of Small Children, Am. J. Dis. Child. **39**:71 (Jan.) 1930.

20. Newburgh, L. H.; Johnston, M. W.; Wiley, F. H.; Sheldon, J. M., and Murrill, W. A.: A Respiration Chamber for Use with Human Subjects, J. Nutrition **13**:193, 1937.

21. Barker, S. B., and Smyth, E.: A Mechanical Pump for the Drawing of Gas Samples, Proc. Soc. Exper. Biol. & Med. **38**:745, 1938.

22. Carpenter, T. M.: Ein Apparat zur Analyse von Gasen aus Respirationskammern für Menschen und Tiere, in Abderhalden, E.: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1933, sect. 55, pt. 13, p. 593.

deviation of 0.003. The rate of ventilation (200 to 300 liters per hour) resulted in carbon dioxide accumulations and oxygen deficits in the outgoing air which ranged from 0.4 to 1.0 per cent and from 0.5 to 1.0 per cent, respectively.

After the sampling, the air was circulated through weighed bottles of sulfuric acid, a process which permitted determination of water vaporized, then through a wash bottle and finally through a wet meter with a volume of 10 liters and a face which permits measurement to the tenth of a liter. The meter was checked periodically against weighed amounts of air.

The accuracy of the technic of measurement was controlled by alcohol checks, the results of which are presented in table 2. For the open circuit the error in oxygen consumption from which heat production was calculated averaged —3.7 per cent, the maximum error being —7.7 per cent. The average errors in carbon dioxide and water were —4.5 and +2.2 per cent, with maximum errors of —7.3 and +6.7 per cent, respectively. The respiratory quotients averaged 0.662 and ranged from 0.65 to 0.67. The average errors in oxygen, carbon dioxide and water for the closed circuit were —3.6, —2.2 and +10.3 per cent, respectively, and the average respiratory quotient was 0.676.

The alcohol lamp used for the open circuit was modeled after the lamp of Newburgh and his co-workers.[20] It consists of an inverted buret of 10 cc. with a side arm in which a glass capillary controls the rate of bubbling and with a glass tip whose size determines the rate of burning. In these observations the heat dissipated averaged 7.2 calories per hour, a level approximating that of the premature infants (table 3).

In the present study no attempt was made to measure the "basal"[23] or standard metabolism of infants, i. e., the caloric expenditure during sleep and after the ingestion of a small meal. Rather, calorimetric observations were begun approximately one hour after the feeding of one eighth or one sixth of the total twenty-four hour caloric intake, which in all instances exceeded maintenance needs. The heat production so measured included the specific dynamic action, or increment of heat due to feeding. Young infants are fed every three to four hours during the day and night because of their relatively limited capacity and greater requirements for food, and their metabolism probably never reaches the true post-absorptive state. The situation is comparable to that of ruminants, and the term "resting metabolism" has been used[24] for the level of heat production which includes the thermogenic effect of food. The energy increment due to activity was determined by comparing periods of repose in the calorimeter with periods of activity.

On the basis of minute to minute activity records inside and outside the calorimeter[16b] collected by specially trained nurses, the energy increments due to activity while in the calorimeter were expanded to cover the twenty-four hour

23. Wetzel (On the Motion of Growth: XVII. Theoretical Foundations, Growth **1**:6, 1937) has emphasized that observations in the calorimeter do not permit fractionation of even this measurement into heat of maintenance, i. e., heat expended in vegetative functions, and heat of growth, or heat expended in the organizational transformations of growth.

24. Brody, S.; Hall, W. C.; Ragsdale, E. C.; Trowbridge, E. A.; Funk, E. M.; Kempster, H. L.; Ashworth, U. S.; Hogan, A. G., and Proctor, R. C.: Growth and Development, with Special Reference to Domestic Animals, Research Bulletin 166, University of Missouri, College of Agriculture, Agricultural Experiment Station, 1932.

TABLE 2.—*Alcohol Checks for Experimental Period (1937-1939) in Terms of Averages per Hour*

Date	Observation No.	Minutes	Number of Periods	Alcohol, Gm.	Water			Carbon Dioxide			Oxygen			Respiratory Quotient (Theoretic 0.667)		Calories, Theoretic
					Theoretic, Gm.	Found, Gm.	Percentage of Error	Theoretic, Gm.	Found, Gm.	Percentage of Error	Theoretic, Gm.	Found, Gm.	Percentage of Error	Found	Error	
Closed Circuit																
Nov. 1	4C2	94	2	0.86	1.151	1.279	+11.1	1.646	1.586	—3.6	1.796	1.684	—6.2	0.685	+0.018	6.10
Nov. 16	4C3	102	2	0.83	.....	.....	.....	1.581	1.565	—1.0	1.725	1.669	—3.2	0.682	+0.015	5.86
Jan. 8	4C4	96	2	1.37	1.827	2.006	+ 9.8	2.613	2.562	—2.0	2.851	2.787	—2.2	0.669	+0.002	9.68
Average				1.02	1.489	1.643	+10.3	1.947	1.904	—2.2	2.124	2.047	—3.6	0.676	+0.009	7.21
Open Circuit								Liters	Liters		Liters	Liters				
March 31	O4	104	2	1.23	1.643	1.622	— 1.3	1.196	1.194	—0.2	1.794	1.794	0	0.666	—0.001	8.70
April 15	O5	95	3	0.80	1.067	1.085	+ 1.7	0.777	0.759	—2.3	1.165	1.167	+0.2	0.650	—0.017	5.65
May 16	O6	60	2	1.71	2.287	2.276	— 0.5	1.665	1.550	—6.9	2.498	2.339	—6.4	0.663	—0.004	12.12
May 27	O7	66	2	1.05	1.398	1.428	+ 2.1	1.018	0.978	—3.9	1.527	1.480	—3.1	0.661	—0.006	7.41
Oct. 3	O16	45	1	0.94	1.241	1.283	+ 3.3	0.919	0.883	—3.9	1.379	1.327	—3.8	0.665	—0.002	6.69
Oct. 27	O17	80	2	0.94	1.234	1.237	+ 0.2	0.913	0.845	—7.3	1.370	1.265	—7.7	0.669	+0.002	6.64
Nov. 29	O18	90	2	0.97	1.277	1.363	+ 6.7	0.945	0.900	—4.6	1.418	1.373	—3.2	0.656	—0.011	6.88
Nov. 30	O19	120	2	0.98	1.292	1.338	+ 3.6	0.956	0.908	—5.0	1.434	1.385	—3.4	0.656	—0.011	6.96
Dec. 2	O20	60	2	0.93	1.229	1.277	+ 3.9	0.909	0.869	—4.4	1.364	1.313	—3.7	0.662	—0.005	6.62
Dec. 13	O21	135	3	0.95	1.252	1.276	+ 1.9	0.926	0.886	—4.3	1.389	1.331	—4.2	0.665	—0.002	6.74
Jan. 27	O22	135	3	0.89	1.170	1.204	+ 2.9	0.865	0.828	—4.3	1.298	1.256	—3.3	0.660	—0.007	6.30
May 17	O25	95	2	0.72	0.947	1.004	+ 6.0	0.701	0.670	—4.4	1.051	1.010	—3.9	0.663	—0.004	5.10
Average				1.01	1.336	1.366	+ 2.2	0.983	0.939	—4.5	1.474	1.420	—3.7	0.662	—0.005	7.15

energy exchange. This was calculated as the sum of the calories expended during sleep, during crying and during wakefulness without crying. The calories expended during feeding were estimated from the latter.

RESULTS

Detailed calorimetric data are presented in table 3 and the expansion of these data on the basis of the twenty-four hour activity records in table 4. In table 5, data on the total energy exchange, including dietary, total catabolic and stored calories, are summarized.

Calorimetric Data.—The total calories measured in the respiratory chamber for observations totaling from four to thirteen hours in infants of different weights and including varying degrees of activity ranged from 5 to 10 calories per hour. The rise in heat production for periods of wakefulness (without crying) above the metabolism during sleep ranged from 6 per cent to 27 per cent for 4 infants (E. C., A. P., R. E. and R. S.) and averaged 15 per cent. In infants R. L. and L. O., whose wakeful periods included crying for 24 and 33 per cent of the time, respectively, the rise above "sleeping" metabolism for such activity averaged 13 per cent. These small rises in heat production accompanying wakefulness are attributable to the slight activity of most premature infants when awake. In occasional calorimetric observations, the details of which have been omitted to conserve space, the oxygen consumption during an interval of sleep beginning one hour after a meal was approximately the same as that in later periods in which the infant was awake and restless, indicating that the thermogenic effects of food and those of mild activity were of similar magnitude.

Crying, on the other hand, was accompanied in all except 2 infants by large increases in metabolism, ranging from 64 to 107 per cent and averaging 90 per cent. Murlin and his co-workers [25] have reported that crying doubles the metabolism in newborn babies.

The infants slept an average of 80 per cent (range 68 to 89 per cent, table 3) of the time spent in the respiratory chamber as compared with 61 per cent for the total twenty-four hours (table 4). This increase in sleeping time in the respiratory chamber has been noted previously for full term infants [16b] and indicates that the infants are not made uncomfortable by their confinement in the chamber. The decreased caloric expenditure due to increased sleeping contributes to the high daily energy balances found when infants were studied in the calorimeter for twenty to twenty-two hours of the day.[15]

The total and nonprotein respiratory quotients ranged from 0.87 to 0.97 and from 0.88 to 1.02 and averaged 0.92 and 0.94, respectively.

25. Murlin, J. R.; Conklin, R. E., and Marsh, M. E.: Energy Metabolism of Normal New-Born Babies with Special Reference to the Influence of Food and of Crying, Am. J. Dis. Child. **29**:1 (Jan.) 1925.

TABLE 3.—*Calorimetric Data in Terms of Averages per Hour*

Subject*	Number of Observations†	Number of Periods	Range in Age, Days	Average Weight, Gm.	Duration of Observation		Percentage of Activity in Total Period‡			Average Rectal Temperature, C.	Chamber		Carbon Dioxide, Liters	Oxygen Liters	Nitrogen in Urine, Gm.	Respiratory Quotient		Calories per Hr.	Rise Above "Sleeping" Metabolism	
											Average Temperature, C.	Average Humidity, %				Total	Non-Protein		Awake %	Crying %
					Hr.	Min.	Asleep	Awake	Crying											
L. O.	4	15	44-74	3378	12	55	89	9	2	37.1	25.33	31	1.929	2.074	0.0473	0.930	0.950	10.17		
					11	29	100	..	..	....	.....	..	.....	.....	.....	.....	.....	9.23		
					1	26	..	67	33	....	.....	..	.....	.....	.....	.....	.....	10.67	16§	
H. L.	3	11	21-33	2760	10	30	92	3	5	37.0	22.76	33	1.613	1.738	0.0603	0.923	0.960	8.50		
					9	57	97	3	..	....	.....	..	.....	.....	.....	.....	.....	8.19		
					..	33	..	..	100	....	.....	..	.....	.....	.....	.....	.....	16.75	..	105
E. C.	4	16	12-25	2392	9	55	79	12	9	36.8	25.14	53	1.305	1.455	0.0433	0.897	0.917	7.07		
					7	51	100	..	..	....	.....	..	.....	.....	.....	.....	.....	6.17		
					1	9	..	100	..	....	.....	..	.....	.....	.....	.....	.....	7.02	14	
					..	55	..	..	100	....	.....	..	.....	.....	.....	.....	.....	11.50	..	86
R. L.	2	5	45-56	2000	4	2	70	24	6	36.9	25.12	51	0.963	1.054	0.0270	0.914	0.933	5.16		
					2	49	100	..	..	....	.....	..	.....	.....	.....	.....	.....	5.01		
					1	13	..	76	24	....	.....	..	.....	.....	.....	.....	.....	5.51	10‖	
M. B.	3	9	25-40	2122	5	8	73	18	9	36.7	27.04	55	1.192	1.300	0.0077	0.917	0.921	6.42		
					4	40	81	19	..	....	.....	..	.....	.....	.....	.....	.....	5.85		
					..	28	..	..	100	....	.....	..	.....	.....	.....	.....	.....	11.64	..	99
A. P.	4	13	10-32	2514	10	5	86	10	4	36.8	27.35	56	1.172	1.343	0.0243	0.873	0.881	6.52		
					8	44	100	..	..	....	.....	..	.....	.....	.....	.....	.....	6.15		
					..	58	..	100	..	....	.....	..	.....	.....	.....	.....	.....	7.03	14	
					..	23	..	..	100	....	.....	..	.....	.....	.....	.....	.....	12.17	..	98
R. E.	4	13	11-27	2378	9	31	89	8	3	36.9	27.39	55	1.270	1.391	0.0479	0.913	0.940	6.78		
					8	31	100	..	..	....	.....	..	.....	.....	.....	.....	.....	6.19		
					..	43	..	100	..	....	.....	..	.....	.....	.....	.....	.....	7.86	27	
					..	17	..	..	100	....	.....	..	.....	.....	.....	.....	.....	12.81	..	107
G. C.	3	10	46-65	1974	7	30	77	18	5	36.4	26.83	48	1.004	1.067	0.0202	0.941	0.958	5.27		
					7	6	81	19	..	....	.....	..	.....	.....	.....	.....	.....	4.95		
					..	24	..	..	100	....	.....	..	.....	.....	.....	.....	.....	8.12	..	64
R. S.	1	6	20	2246	4	10	68	23	9	36.5	27.68	50	1.150	1.181	0.0425	0.974	1.019	5.84		
					2	49	100	..	..	....	.....	..	.....	.....	.....	.....	.....	5.53		
					..	58	..	100	..	....	.....	..	.....	.....	.....	.....	.....	5.88	6	
					..	23	..	..	100	....	.....	..	.....	.....	.....	.....	.....	9.26	..	67

* No calorimetric observations were made on infants N. O. and E. M.; the total calories were predicted from the insensible perspiration.

† In order to conserve space the detailed calorimetric data for individual observations are not recorded.

‡ For each infant the top line gives the energy exchange for the entire time in the respiration chamber; the second line that for periods in which sleep occupied from 80 to 100 per cent of the time; the remaining lines that for wakefulness and crying as indicated in the chart.

§ Includes crying 33 per cent of the time.

‖ Includes weak crying 24 per cent of the time.

TABLE 4.—*Daily Behavior and Caloric Expenditure in Terms of Twenty-Four Hours*

Subject	Days of Study	Average Age, Days	Average Weight, Gm.	Average Surface Area, Square Meters*	Daily Activity Record: Minutes: Asleep	Minutes: Awake: Quiet	Minutes: Awake: Restless	Minutes: Awake: Feeding	Minutes: Awake: Crying	Percentage of Total: Asleep	Percentage of Total: Awake: Quiet	Percentage of Total: Awake: Restless	Percentage of Total: Awake: Feeding	Percentage of Total: Awake: Crying	Total Calories Expended
N. O.	12	24	2106	0.161	757	219	276	91	97	53	15	19	6	7	132†
E. M.	11	30	2262	0.170	780	175	241	107	137	54	12	17	7	10	163†
L. O.	9	37	2641	0.191	806	253	150	79	152	56	18	10	5	11	185
H. L.	10	29	2720	0.195	816	116	271	81	156	56	8	19	6	11	210
E. C.	11	21	2442	0.180	805	171	195	81	188	55	12	14	6	13	181
R. L.	20	53	2101	0.161	989	124	176	126	25	68	9	12	9	2	133
M. B.	24	31	2071	0.159	996	77	211	83	73	69	5	15	6	5	144
A. P.	9	22	2551	0.186	900	102	230	127	81	62	7	16	9	6	166
R. E.	9	15	2217	0.168	933	78	193	111	125	65	5	13	8	9	161
G. C.	9	51	1850	0.146	871	165	241	133	30	61	11	17	9	2	114
R. S.	6	20	2277	0.171	972	75	222	71	100	68	5	15	5	7	143
Total average	12	30	2294	0.172	875	141	219	99	106	61	10	15	7	7	157
14 Full term infants	7 months (3-11 months)‡									50 (39-44)	38 (28-52)	7 (1-13)	3 (1-8)	3 (2-7)	

* The surface area was calculated from the formula given by A. D. Klein and R. E. Scammon (Relations Between Surface Area Weight and Length of the Human Body in Prenatal Life, Proc. Soc. Exper. Biol. & Med. **27**: 456, 1930): 5.188 wt. 0.750.

† The values were calculated from the insensible perspiration.

‡ The figures in parentheses indicate range.

The average total and nonprotein respiratory quotients for full term infants studied under similar dietary conditions were 0.88 and 0.90, respectively.[15] If one accepts the respiratory quotient as an index of the metabolic mixture, carbohydrate supplied from 61 to 100 per cent and averaged 81 per cent of the nonprotein calories.

Daily Behavior and the Equivalent Caloric Expenditure.—The time spent in sleep by the 11 infants whose ages during observations varied from 15 to 53 days averaged fourteen and a half hours (range twelve and a half to sixteen and a half hours). The time spent in quiet wakefulness averaged two and one-third hours and in restless wakefulness three and two-thirds hours. Feeding time (6 to 8 feedings per day) averaged one and two-thirds hours (range one and one-sixth to two and one-fourth hours) and crying time averaged one and three-fourths hours (range one-half to three hours). The premature infants slept 61 per cent of the time, as compared with 50 per cent for a group of 14 older full term infants under similar conditions of environment and nursing care.[26] Crying and feeding each occupied 7 per cent of the day, as compared with 3 per cent for each in the full term group. Wakefulness, both quiet and restless, represented 45 per cent of the day for full term infants, as compared with 25 per cent for the premature infants. The smaller figure for wakefulness in the premature infants indicates a simpler existence, in which sleep, feeding and crying (usually for food) accounted for 75 per cent of the total twenty-four hours, as compared with 56 per cent for the same quotas for full term infants.

Total Catabolism.—The total catabolism (calories expended per twenty-four hours in basal metabolism, in thermogenic effect of food and in activity) ranged from 114 to 210 and averaged 157 calories per twenty-four hours (table 4).

The total caloric expenditure varied for individual infants from 61 to 77 and averaged 68 calories per kilogram per twenty-four hours. Rubner and Langstein [3] found an average of 71 calories per kilogram and a range of 69 to 76 in their 4 observations on 2 infants. These small variations are related to the limited activity indulged in by premature infants (table 4).

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26. In assessing the significance of these records of activity it is important to note the conditions under which they were collected. First, all infants were on a mattress which permitted collection of urine and stools. (The records of the activity of 3 premature infants during the period when no collections were being made showed no appreciable difference from their records of activity when collections were being made. The activity of premature infants is apparently not hampered by the artificial restraints during periods when collections are made.) Second, nurses were in constant attendance on these infants, and while they could mollify older, full term infants by such methods as the use of rattles, the chief cause of the crying of premature infants was hunger, which could be eliminated only by increasing the intake of food.

The average of 68 calories is only 18 per cent above the previously reported "basal" level of 58 calories per kilogram per twenty-four hours.[17] In a group of older, full term infants the average total catabolism under comparable conditions was 82 calories per kilogram,[9] an increase of about 40 per cent above the basal rate. The divergence in total twenty-four hour catabolism between premature and full term infants would be even greater if referred to surface area, because of the larger surface of the smaller infants. Since heat loss is conditioned by the total radiating surface, among other factors, the tendency of premature infants to subnormal body temperatures is increased by this disproportion between total heat production and surface area.

TABLE 5.—*Total Daily Energy Exchange, Summary of Results*

Subject	Average Weight, Gm.	Daily Weight Gain, Gm. per Kg.	Diet Calories per Kg.	Total Catabolism Calories per Kg.	Feces		Energy Balance		
					Calories per Kg.	Percentage of Dietary Calories	Calories per Kg.	Percentage of Dietary Calories	Calories per Gm. of Weight Gain
N. O.	2106	14.9	114	62.9	16.3	14	34.8	31	2.3
E. M.	2262	14.0	113	71.9	27.0	24	14.1	12	1.0
L. O.	2641	17.9	123	70.1	9.7	8	43.2	35	2.4
H. L.	2720	9.8	135	77.1	22.2	16	35.7	26	3.6
E. C.	2442	14.4	126	74.0	9.4	7	42.6	34	3.0
R. L.	2101	18.4	118	63.1	14.1	12	40.8	35	2.2
M. B.	2071	14.8	144	69.6	25.1	17	49.3	34	3.3
A. P.	2551	13.5	104	65.0	12.1	12	26.9	26	2.0
R. E.	2217	18.6	116	72.7	19.0	16	24.3	21	1.3
G. C.	1850	21.1	118	61.4	31.9	27	24.7	21	1.2
R. S.	2277	20.4	107	62.8	11.9	11	31.8	30	1.6
Average	2294	16.2	120	68.2	18.1	15	33.5	28	2.3*

* $\frac{\text{Total calories stored}}{\text{Total weight gain, Gm.}} = \frac{10{,}485}{4{,}622} = 2.3$

Caloric Loss in Feces.—The calories lost in the feces by individual infants ranged from 9 to 32 and averaged 18 calories per kilogram per twenty-four hours. This is approximately double the fecal loss (5 to 10 calories) generally accepted[27] for full term infants. Of more significance than this twofold increase in the average is the finding that in 4 infants the loss was between 22 and 32 calories per kilogram, an amount which might leave inadequate calories for weight gain on customary diets. The major portion of this fecal loss was due to greater excretion of fat. The effect of varying the quality and quantity of fat ingested on fat absorption and weight gain is at present being studied.

2/. (*a*) Talbot, F.: The Caloric Requirements of Normal Infants and Children from Birth to Puberty, Am. J. Dis. Child. **18**:229 (Oct.) 1919. (*b*) Holt, L. E., and Fales, H. L.: The Food Requirements of Children: I. Total Caloric Requirements, ibid. **21**:1 (Jan.) 1921. (*c*) Fleming, G. B.: The Theoretical Food Requirements of Infants, Brit. M. J. **2**:1093, 1924.

Energy Balance.—The feeding of diets containing an average of 120 calories per kilogram (range 104 to 144) permitted an average weight gain of 16 Gm. per kilogram (range 10 to 21) and an average storage of energy of 34 calories per kilogram (range 14 to 49). In this group of premature infants the increased loss of calories in feces as compared with that in full term infants was counterbalanced by the lower total catabolism, so that diets of caloric intakes equivalent to those fed full term infants permitted satisfactory weight gains. Methods of feeding designed to decrease the fecal loss might permit satisfactory weight gains with total caloric intakes of even less than 120 calories per kilogram.

The weight gain for the entire group of infants for one hundred and thirty days totaled 4,622 Gm., and the calories stored totaled 10,485, so that 2.3 calories was stored for each gram of weight gain (table 5). This compares favorably with the average equivalent of 2.7 calories per gram of weight gain found by Levine in his twenty-four hour calorimetric observations on full term infants,[15] and the value of 2.9 calculated from the observations of Rubner and Langstein [8] for 2 premature infants. The theoretic caloric equivalent of weight gain ($P \times 5.65$, $F \times 9.3$), calculated on the assumption that 13 per cent of the weight gain is protein, 60 to 80 per cent is water and the remainder is fat, ranged from 3.2 to 1.4 calories per gram; with an assumed average composition of 13 per cent protein, 65 per cent water and 22 per cent fat, it equaled 2.7 calories per gram. (The value of 1.72 calories per gram given by Lusk [28] was calculated by Rubner [29] from carcass analyses of different animals.) The calculated figures compared favorably with the results actually obtained, the range of the latter being from 1.0 to 3.6 calories stored per gram of weight gain and the average 2.3. This close agreement between theoretic and found caloric equivalents of the weight gain serves as a check on the reliability of the methods used and affords a rough measure of the excess of dietary calories above maintenance required for a given weight gain.

COMMENT

The finding that diets containing an average of 120 calories per kilogram of body weight per day covered the energy requirements of a group of thriving premature infants prompted closer scrutiny of the practice of feeding between 150 and 200 calories per kilogram. Such practice is based partly on the acceptance of maximal rates of weight gain as optimal and partly on the opinion that premature infants require

28. Lusk, G.: The Science of Nutrition, ed. 4, Philadelphia, W. B. Saunders Company, 1928, p. 567.

29. Rubner, M.: Das Wachstumproblem und die Lebensdauer des Menschen und einiger Säugetiere vom energetischen Standpunkt ausbetrachtet, Arch. f. Hyg. **66**:147, 1908.

a greater caloric intake than do full term infants because of more rapid rate of growth.

What constitutes optimal growth for the premature infant is not yet established, but it seems reasonable to accept the rate of weight gain in utero as one criterion of such growth. The latter may be calculated from the data of Streeter [30] and Scammon and Calkins [31] as approximately 15 Gm. per kilogram per day for infants of 1,000 Gm. and 10 Gm. per kilogram for infants of 2,000 Gm. Adequate energy for such weight gains may be supplied by giving approximately 20 to 40 calories per kilogram per twenty-four hours above maintenance requirements. In these observations a total caloric intake averaging 120 calories per kilogram covered all the energy needs and permitted a gain in weight of 16 Gm. per kilogram, as compared with the theoretic optimal gain of 10 Gm. per kilogram per day for the group of infants studied (table 5).

The statement that premature infants require more calories than full term infants because of their greater rate of growth does not seem valid. The slightly greater amount of energy stored incident to optimal gains of 10 to 15 rather than 8 to 10 Gm. of tissue per kilogram per day hardly serves as an excuse for feeding diets that are from 100 to 150 per cent above the maintenance requirements. Without discussing in detail the large variety and interrelationships of endogenous and exogenous factors [32] which influence growth, it seems reasonable to postulate that high caloric intakes permit rapid weight gains, rather than the converse, that a high rate of growth requires a high caloric intake. (The desirability of distinguishing between weight gain and growth [33] will not be discussed here.)

The finding that an average of 120 calories per kilogram per twenty-four hours covered the maintenance energy requirements of the small group of premature infants studied and provided adequate surplus energy for weight gain does not lessen the necessity for individualizing the feeding of each infant. Individual variations in "basal" metabolism,[17] variations in metabolism due to age,[17] environmental temperature [34] and activity, particularly in crying due to hunger, and variations in ability

30. Streeter, G. L.: Sitting Height, Head Size, Foot Length and Menstrual Age of the Human Embryo, Contrib. Embryol. **11**:145, 1920.

31. Scammon, R. E., and Calkins, L. A.: Growth in the Fetal Period, Minneapolis, University of Minnesota Press, 1929.

32. Brody, S.: Factors Influencing the Apparent Energetic Efficiency of Productive Processes in Farm Animals, J. Nutrition **17**:235, 1939.

33. Hammett, F. S.: Nutrition vs. Growth, Science **86**:50, 1937.

34. Mordhorst, H.: Ueber die chemische Wärmeregulation frühgeborener Säuglinge, Monatschr. f. Kinderh. **55**:174, 1932.

to absorb fat may all influence the total energy expenditure for maintenance. It is conceivable that during the first two weeks of life, when the basal metabolic rate [35] and activity are both low, or under conditions such as the use of diets in which fat supplies less than the customary 35 to 50 per cent of calories,[36] even lower caloric intakes may permit satisfactory gains in weight.

The routine feeding of high caloric intakes (150 to 200 calories per kilogram) may lead to one of two possibilities: First, the infant's tolerance for food may be exceeded and a gastrointestinal disorder result. The premature infant with his poor sucking ability, defective appetite and faulty absorption of fat [37] is notoriously disposed to such disorders, particularly in the presence of fever, infections and high environmental temperatures. Second a rapid gain in weight may ensue, a result of doubtful value in view of the opinion [38] that the tendency to rickets and anemia is accentuated by an excessive rate of growth. The inverse relationship between mortality rate and birth weight in premature infants sometimes leads to the too ready belief that the sooner an infant of 1,500 Gm. is converted into an infant of 3,000 Gm., the better is his prognosis. The rate of intrauterine conversion might well serve as one guide in extrauterine feeding.

The tendency of premature infants to have subnormal body temperatures and their lower basal metabolic rate when referred to surface area [35] as compared with that of full term infants have led to the belief that premature infants have a defect in oxidative capacities. Caution must be used in the acceptance of such conclusions on the basis of data so far available. Factors which militate against a simple comparison of basal metabolic rates are: possible differences in response of premature and

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35. (*a*) Talbot, F. B.; Sisson, W. R.; Moriarty, M. E., and Dalrymple, A. J.: The Basal Metabolism of Prematurity: Metabolism Findings in Twenty-One Premature Infants, Am. J. Dis. Child. **26**:29 (July) 1923. (*b*) Marsh, M. E., and Murlin, J. R.: Energy Metabolism of Premature and Undersized Infants, ibid. **30**:310 (Sept.) 1925. (*c*) Schadow, H.: Der Betriebstoffwechsel und Kalorienbedarf frühgeborener Säuglinge, Jahrb. f. Kinderh. **136**:1, 1932. (*d*) Gordon and Levine.[17]

36. Powers, G. F.: Comparison and Interpretation on a Caloric Basis of the Milk Mixtures Used in Infant Feeding, Am. J. Dis. Child. **30**:453 (Oct.) 1925.

37. (*a*) Tidwell, H. C.; Holt, L. E., Jr.; Farrow, H. L., and Neale, S.: Studies in Fat Metabolism: II. Fat Absorption in Premature Infants and Twin, J. Pediat. **6**:481, 1935. (*b*) Rubner and Langstein.[3]

38. (*a*) Eliot, M. M.: The Control of Rickets, J. A. M. A. **85**:656 (Aug. 29) 1925. (*b*) Park, E. A.: The Etiology of Rickets, Physiol. Rev. **3**:106, 1923. (*c*) Davidson, L. T., and Merritt, K. K.: Viosterol in the Prophylaxis of Rickets in Premature Infants: Clinical, Chemical and Roentgenologic Observations, Am. J. Dis. Child. **48**:281 (Aug.) 1934. (*d*) Ylppö, A.: Zur Physiologie Klinik und zum Schicksal der Frühgeborenen, Ztschr. f. Kinderh. **24**:1, 1919.

of full term infants to the same environmental temperature; differences in level of previous feeding, particularly the excess of protein and caloric intake over maintenance needs; differences in rate of growth and therefore in "heat of growth," [23] and differences in composition of the body. Subjects whose oxidative rates might be comparable are premature infants and marasmic full term infants of the same postnatal ages, kept at high environmental temperatures (thermal equilibrium) and gaining at approximately the same rate, the latter being a guide to both heat of growth and excess of food over maintenance metabolic needs.

If one accepts as a measure of general oxidative efficiency the ability of the growing organism to transform food energy into tissue, then paradoxically the lower total catabolism of the premature infant permits a greater storage of energy with a given absorbable caloric intake than occurs in the full term infant. Specific metabolic peculiarities may exist of course, as is indicated by the defect in fat absorption and the incomplete breakdown of certain amino acids.[39] The dominant cause of the tendency of premature infants to subnormal temperatures, however, may be their large surface area and lack of fatty insulation, rather than defective oxidation, for one might reasonably expect an animal of this size to have a fur coat.

SUMMARY

The total twenty-four hour energy metabolism of 11 healthy premature male infants who at the onset of observations ranged in age from 10 to 44 days and in weight from 1,543 to 2,564 Gm. was determined for a period totaling one hundred and thirty days under customary conditions of diet and environment. Analyses of diet and excreta combined with calorimetric observations over short periods and twenty-four hour minute to minute activity records permitted determination of the total catabolism, fecal calories and energy balance.

The total catabolism ("basal" metabolism, specific dynamic action or thermogenic effect of food and activity) averaged 68 calories per kilogram per twenty-four hours and ranged from 61 to 77. This average figure is only 18 per cent above the previously reported basal level of 58 calories.

The fecal loss averaged 18 calories per kilogram per twenty-four hours and ranged from 9 to 32 calories. The average figure is approximately double the fecal loss accepted for full term infants. The greater caloric loss in feces was due to greater excretion of fat.

The feeding of diets averaging 120 calories (range 104 to 144) permitted storage of 34 (range 14 to 49) calories per kilogram per

39. Levine, S. Z.; Marples, E., and Gordon, H. H.: A Defect in the Metabolism of Aromatic Amino Acids in Premature Infants: The Role of Vitamin C, Science **90**:620, 1939; Amino Acid Metabolism in Premature Infants, to be published.

twenty-four hours. The average daily weight gain was 37 Gm. (16 Gm. per kilogram), and the average energy balance was 2.3 calories per gram of weight gain.

Factors which may serve as a rational basis for individualization in the feeding of premature infants are discussed.

CONCLUSIONS

Diets which supply approximately 120 calories per kilogram per twenty-four hours may be considered adequate under customary environmental conditions to cover the maintenance energy requirements and to provide a surplus for satisfactory weight gain in healthy premature infants more than 2 weeks of age.

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DISTORTED SPEECH IN YOUNG CHILDREN

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Considerable attention has been paid to such speech defects of childhood as stuttering and dyslalia resulting from cleft palate and other physical malformations. On the other hand, relatively little attention has been given to articulatory defects in children, although such defects are seen frequently in speech clinics. The children often exhibit disorders of articulation so extreme as to cause their speech to be practically unintelligible, and yet they show no obvious physical defect which might account for the speech distortion. In fact, they appear to be alert, to have histories of normal development and to comprehend spoken language. The only abnormality which they present is a failure of good speech to develop. The present study is an investigation of the factor or factors which are associated with this distorted or delayed speech in young children.

The general problem may be approached from a number of points of view. First, the development of symbolic language and normal articulation may be regarded as dependent on the stage of the child's neural maturation. This in turn may be correlated with the level of the child's physical and psychologic development. In an earlier article two of us [1] pointed out the probable relation between the process of myelinization and the speech status of the developing boy and girl. In the second place, the delayed speech may be attributed to such environmental factors as unfavorable parental attitude, which may in turn produce unfavorable reactions in the child, i. e., dependence, negativism or retarded social adaptation. Finally, pathologic conditions may be operative; for example, there may be an endocrine disorder or a hearing defect. Indeed, any or all of these conditions may operate coincidentally to cause delayed or distorted speech. Hence it was deemed necessary to include

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Gertrude Miller, M.A., assisted in the speech testing and in the preparation of this paper.

1. Karlin, I. W., and Kennedy, L.: Delay in the Development of Speech, Am. J. Dis. Child. **51**:1138 (May) 1936.

both developmental functions and independent factors in the investigation, with a view to identifying as nearly as is possible in an exploratory study the underlying cause of distorted speech in the children available for this study.

The investigation consisted of (1) selection of the subjects, (2) speech analyses, (3) physical and neurologic examination, which included roentgenographic studies of the centers of ossification in the wrist and a determination of the content of cholesterol in the blood, (4) psychologic tests and (5) audiometric tests.

SELECTION OF SUBJECTS

At the time this study was undertaken there were 9 children in the speech clinic of the Brooklyn Jewish Hospital who, because of their markedly distorted speech, had been referred for diagnosis and treatment. They ranged in age from 3 years 6 months to 8 years 3 months. In order to make group comparisons, 3 children were eliminated; 2 of them were under 5 years of age, and the third was excluded because it was impossible to obtain satisfactory test results for him. Six children were retained, 3 boys and 3 girls between 6 and 8 years of age. These children will be referred to in this study as the experimental group. The cases of the 3 children who were eliminated from the experimental group will be presented in a separate section.

In addition to this experimental group a control group consisting of 6 children paired individually with the 6 children of the experimental group was formed. The controls were from the same group of hospital patients, clients of the free clinic. They had recovered from the ailments for which they had been treated and were available on the days on which they returned for a follow-up examination. In a measure, then, the two groups had comparable socioeconomic status and local experiences. Each child in the delayed speech group was paired with a normal-speaking control. The members of each pair were matched in age and sex and were approximately matched in mental level, according to the Stanford-Binet intelligence scale. No attempt was made to match them with respect to linguistic background. Some of the children heard English and Italian in their homes, some English and Yiddish and some only English. The children acting as controls were given the entire series of tests and examinations which the experimental group had been given.

SPEECH ANALYSES

The speech of the children of the experimental group was appraised and analyzed. In the first place, three judges experienced in recognizing phonetic lapses ranked the 6 subjects in the order of their approximation to normal speech; i. e., the speech of subject 1 was the least mutilated,

whereas the speech of subject 6 was the most difficult to understand. Secondly, each child in the experimental group was given the first two articulation tests, one in reproduction and the other in speed, of the Monroe reading aptitude tests.[2] Lastly, two tests for more simple articulatory performances were devised. One measured the subject's control over the muscles of articulation; the other, his skill in imitating sound units.

In the test for control of the articulatory muscles, each subject was observed to determine whether or not he was able to make the basic articulatory adjustments of the palate, tongue and lips required to produce the English sound units ordinarily mastered by children of comparable age. Perfect performance of the three articulators yielded a score of 100 points. Forty points each were given for normal velar movements and normal tongue movements, and 20 points were given for normal lip movements. The velum was observed in order to determine whether or not it closed the nasal port. The tongue was tested for the following adjustments: (1) elevation and spreading, as in "t"; (2) pulling down, as in "ta"; (3) protruding, as in "th"; (4) breaking contact between side of tongue and teeth, as in "l," and (5) drawing back, as in "k." The following lip adjustments were observed: (1) rounding, as in "u" (oo); (2) protruding, as in "sh"; (3) spreading, as in "i" (ee); (4) contact between upper teeth and lower lip, as in "f-v," and (5) forming central orifice, as in "th."

In the test for the reproduction of sound units, the subjects were asked to imitate the examiner's sound units when they were produced as isolated sounds. Each sound unit was arbitrarily assigned a numerical value, the consonants totalling 60 and the vowels and diphthongs 40. Thus, any subject who imitated the English sounds accurately obtained a score of 100. The value assigned to any sound corresponded roughly to its frequency of occurrence in commonly used words[3]: "t" was given a value of 11; "n," 7; "p," 4; "sh," 1. The ratings of the subjects in these tests appear in table 1.

An examination of the table reveals that there is a general relation between the relative rating in speech and the age. The 3 oldest children ranked the three highest in speech, and the youngest ranked the lowest, the child who ranked lowest of all being the youngest.

We were dependent on the mother for information regarding the age at the onset of speech. The mother of subject 2 reported that the boy babbled at the age of 2, but that she was unable to comprehend his

2. Monroe, M.: Primary Form Aptitude Tests, Boston, Houghton Mifflin Company, 1935.

3. Fletcher, H.: Speech and Hearing, New York, D. Van Nostrand Company, Inc., 1929, p. 84.

speech until he reached the age of 5. This subject is the twin brother of a girl whose speech is normal in all respects. It is noteworthy that the onset of speech occurred at a considerably later age among the boys than among the girls. This is consistent with the observations of many other investigators.

In the Monroe reproduction and speech tests all the subjects rated exceedingly low, the highest reaching the sixth percentile and 2 subjects reaching only the first percentile. Since the subjects were selected because of articulatory speech defects, it is to be expected that they would be below average on an articulation test. The Monroe norm is based on the combined rating of reproduction and speed; hence we were unable to evaluate the factor of speed singly. We feel justified, however, in inferring that the exceedingly low combined score is due in some

TABLE 1.—*Results of the Speech Test of the Patients in the Experimental Group*

Subject*	Sex	Age		I. Q.	Percentage of Hearing Loss		Age at Onset of Speech	Monroe Score (Percentile)	Articulatory Movements, Score	Reproduction of Sound Units	
		Yr.	Mo.		Right Ear	Left Ear				Score	Defective Sounds
1	♂	6	2	90	13.6	9.6	3	6	100	96	th
2	♂	8	3	107	20.8	19.2	5	1	100	92	r, sh
3	♀	7	5	90	22.4	15.2	1½	1	60	85	r, s
4	♀	5	7	89	4.8	20.8	2½	3	100	92	r, sh
5	♀	6	0	71 †	29.6	20.0	2	2	90	75	sh, ng, k, r, s, th
6	♂	5	2	84	33.6	36.0	3	5	100	78	sh, k, th, r, s

* The rank was based on the general intelligibility of the child's speech.
† The examiner feels that the I. Q. is spuriously low and that an adequate retest would yield a somewhat higher I. Q. for this subject.

measure to the factor of speed. In our opinion, these children are unable to perform repetitive movements of the articulatory muscles with the speed required for normal speech. Studies by Travis [4] and by West [5] showed a similar slowness of diadokokinesis in stutterers. Four of the 6 subjects were able to make all the basic articulatory movements necessary for producing English sound units. Subject 1 showed socially infantile speech; i. e., his speech lacked vigor, conviction and attention. Subject 3 was unable to elevate the palate; hence her speech was characterized by extreme nasality. All the vowels were nasalized; many of the consonants resembled the distorted sounds present in the speech of persons with cleft palate. Subject 6 was unable to protrude the tongue, as is required in the production of the "th" sound.

4. Travis, L.: Speech Pathology, New York, D. Appleton and Company, 1931, pp. 223-226.

5. West, R.: A Neurological Test for Stutterers, J. Neurol. & Psychopath. **10**:114, 1929.

All the subjects were able to imitate isolated sound units surprisingly well, although none was able to combine them into words. The sounds which the children were unable to reproduce were "r" (5 cases), "sh" (4 cases), "s" (3 cases), "th" (3 cases) and "k" (2 cases) and "ng" (1 case). These are the troublesome consonants, because of the delicate adjustments required for their production owing to their relatively low phonetic power [3] and because, in the case of "k" and "ng," it is impossible for the chlid to get visual cues to aid him in producing the sound. Articulation of the last group of sounds ("k," "ng") is especially defective among children who are hard of hearing. Interestingly enough, the 2 patients who failed to imitate these sounds presented the greatest hearing loss. The subject who lacks the greatest number (six) of consonant sounds is the child having the lowest Binet score.[6] She is also one of the youngest children and has a considerable hearing loss.

PHYSICAL AND NEUROLOGIC EXAMINATION

All the subjects of this investigation were given complete physical and neurologic examinations. The children of the experimental group had been referred to the hospital only because of distorted speech. The developmental history was normal in regard to teething, sitting and walking. The mothers were unable to tell the exact date of the beginning of speech because of its similarity to the earlier babbling. The physical examination revealed abnormalities in several patients of the experimental group. Subject 1 presented mild secondary anemia and markedly delayed maturation in the centers of ossification (table 2). Subject 3 presented cleft palate speech without cleft palate; her palatal reflex was markedly diminished. Subject 4 had an abnormally shaped head, a markedly flattened occipital region and an increase in the vertical diameter; the presence of a turret skull was confirmed by roentgenogram. Subject 5 was extremely obese. The endocrine clinic reported that her obesity was probably exogenous, although it might possibly be of pituitary origin. Her basal metabolic rate was + 4.

Because the speech of these children resembled, to a certain extent, the speech of children much younger, we attempted to determine whether or not there was subclinical evidence of delayed growth. Since the thyroid is a dominant factor in growth production and since hypothyroidism is manifested by a delay in maturation of the centers ot ossification and by a high content of cholesterol in the blood, roentgenograms of the wrists were taken and determinations of the blood cholesterol were made. The results are given in table 2.

6. See footnote† to table 1.

Of the 9 patients with distorted speech, 3 showed delay in the development of the centers of ossification. Of the 6 controls, 2 showed retardation. All the blood cholesterol values were within normal limits, although several were in the higher brackets (250 mg.).

It is interesting to note that in the experimental group the child who showed the most marked delay in the maturation of the centers at the wrists (fig. 1) had the most intelligible speech, according to the speech analysis. The striking feature in connection with his speech, however, was a lack of desire to communicate with others and a general meagerness of expression. This child is being treated with thyroid.

TABLE 2.—*Skeletal Maturity and Content of Cholesterol in Blood of Six Pairs of Children, One with Distorted and the Other with Normal Speech, and of Three Additional Children with Distorted Speech*

Subject* and Control	Sex	Age		Ossification of Wrists		Blood Cholesterol	
		Yr.	Mo.	Subject	Control	Subject	Control
1	♂	6	2	Markedly delayed	Delayed	176	174
2	♂	8	3	Normal	Normal	202	234
3	♀	7	5	Normal	Normal	190	190
4	♀	5	7	Normal	Normal	230	250
5	♀	6	0	Normal	Normal	194	176
6	♂	5	2	Delayed	Delayed	196	190
Additional Patients Excluded from Experimental Group							
S. Y.	♂	6	4	Delayed	.....	191	
E. T.	♀	3	6	Normal	.....	250	
A. R.	♂	4	7	Normal	.....	257	

* The rank among the children of the experimental group was based on the general intelligibility of the child's speech. All the controls had normal speech.

PSYCHOLOGIC TESTS

It has been mentioned previously that the 9 children with delayed speech in the clinic group and the children of the control group were given a series of psychologic tests. This series consisted of the Stanford-Binet test (1937 revision, form L), the Pintener-Patterson performance series and the Vineland social maturity scale. The results, in terms of median mental age achieved by the experimental and the control group, are presented in table 3. A real effort was made to have the controls match the experimental patients in Stanford-Binet mental age; exact matches, however, were not always found. Consequently, the median mental age of the control group is 5 years 9 months, as contrasted with the median of 5 years 7 months achieved by the experimental group. Correspondingly, the median intelligence quotient of the experimental group was 89, while the median of the controls was 93. Although the

children with delayed speech appear to be slightly slower than average in mental development, according to the Stanford-Binet test for mental ability, no definite conclusions in this respect are justified. The distorted speech may, directly or indirectly, provide a language handicap on a test so prominently verbal as the Stanford-Binet form L; or, if defective hearing contributes to the child's speech difficulties, then the same hearing defect may prevent him from understanding the directions adequately in the test situation. Finally, in view of the few children studied, any number of selective factors may be operating to place duller children in the clinic group.

Even in such a small group, however, some evidence of accelerated or retarded functioning can be derived from a comparison of the type of

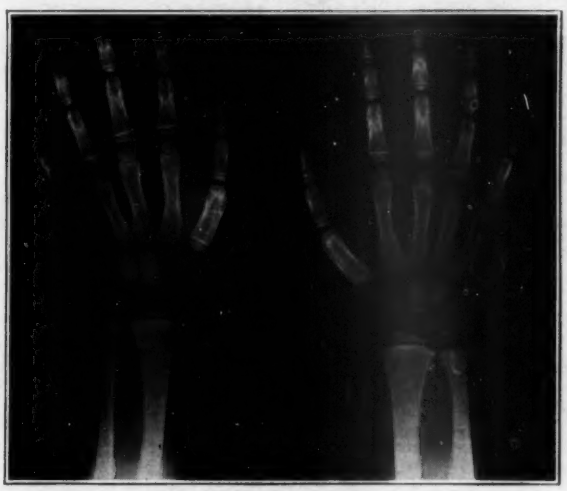

Fig. 1.—Roentgenogram of the wrists of a boy 6 years 2 months of age (subject 1, table 2), showing three centers of ossification, the third in each instance being only faintly discernible. The findings represent a marked delay in the development of ossification and suggest hypothyroidism.

performance shown by the members of the experimental group and that shown by the controls in the varied tests used in this study. For example, if children with retarded speech are considerably handicapped in language usage, then when compared with controls matched on the basis of a predominantly verbal intelligence test they should exhibit relatively superior scores on performance tests. This is not always the result. It is apparent in table 3 and in figure 2 that in some tests, such as the first Manikin and Healy picture completion test, the children with distorted speech did show median performances which were superior to the performances shown by their matched controls. On the Knox cube

test, however, there was no difference in median performance between the two groups. On the other two performance tests, i. e., the Seguin formboard and the mare and foal test, quite the reverse was found; the controls were much better than the children of the experimental group. The special superiority of the controls on these speed tests accounts for the degree to which they surpass the patients with delayed speech when the composite performance median is obtained. So few children of the experimental group were able to score at all on the more difficult tasks of the Pintner-Patterson series that the results for only the easier performance tests are tabulated. The difference found between the groups on the Vineland social maturity scale is only what may be readily

TABLE 3.—*Results of Psychologic Tests Administered to Children with Distorted Speech and to Their Matched Controls*

	Experimental Group		Control Group		Number of Children in Each Group
Name of Measure	Median Age in Yr. and Mo.	Range	Median Age in Yr. and Mo.	Range	
Chronologic Age.....	6 - 3	5 - 2 to 7 -11	6 - 3	5 - 0 to 7 - 5	6
Stanford-Binet (L)....	5 - 7	4 - 2 to 8 - 6	5 - 9	4 - 1 to 7 - 0	6
Seguin Formboard....	5 - 9	5 - 5 to 6 - 3	6 - 3	6 - 3 to 8 - 6	5*
Mare and Foal.....	Less than 5 - 0	Less than 5 - 0 to 5 -11	6 - 6	5 - 4 to 11 - 5	5
Manikin.....	7 - 6	5 - 6 to 8 - 6	6 - 6	Failed to 8 - 6	5
Knox Cubes.....	6 - 2	5 - 6 to 6 - 6	6 - 0	4 - 0 to 6 - 6	5
Healy P. C. I.....	5 - 6	4 - 6 to 10 - 9	4 -11	4 - 6 to 6 - 7	5
Composite of Performance Tests (Medians)..	5 - 9	5 -11 to 6 - 4	6 - 3	4 - 8 to 7 - 8	5
Vineland Social Scale, S. A.	5 -10	4 - 8 to 7 - 8	5 -11	4 -11 to 7 - 9	6

* Because of the persistent absences from the clinic one child with delayed speech failed to complete the performance series. His matched control has been dropped in the performance comparisons.

predicted with the existing difference in the Stanford-Binet performance, because of the correlation between the two tests.

Several interesting suggestions can be obtained from a further study of these results:

1. Since the children with distorted speech are slightly poorer on the combined performance measure than on the Stanford-Binet, and since the controls are considerably better on the combined performance than on the Stanford-Binet, there is no indication that the children with delayed speech were excessively handicapped in the Stanford-Binet test because of their lack of verbal facility.

2. The two performance tests in which the children with delayed speech excelled demanded logical organization of visually attractive materials with no time limitation.

3. The two performance tests on which the children with delayed speech were completely outstripped by the control group were timed tests in which the greatest premium was placed on motor speed. It is important to note, also, that these two tests are so simple that they can be administered to children speaking only a foreign language or even to deaf-mutes. Thus, there is scarcely a possibility that the poor showing made by the children of the experimental group in this study resulted from failure to understand the test directions.

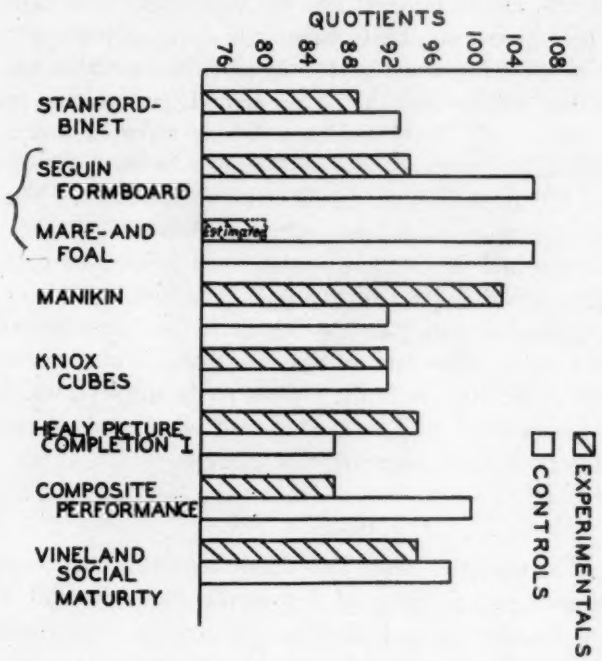

Fig. 2.—Chart comparing the median quotients on the psychologic tests obtained by the experimental group with the median quotients obtained by the control group.

It cannot be repeated too often that these results are merely suggestive and are not statistically reliable, since the groups are so small. However, should the finding that children with markedly delayed speech show poor ability at tasks requiring motor speed be confirmed, it would be of great interest. Only three studies relating in any way to this problem have come to the attention of the authors. Arps [7] found that the motor efficiency of grade school children with speech defects was poorer than that of other children. This is not direct confirmation of

7. Arps, W.: Ueber die motorische Leistungsfähigkeit bei Grundschulkindern der Sprachheil-, Volks-und Hilfsschule, Hamburg, Lehrerzing **13**:597, 1934.

our observations, for the children of his group were slightly older than those in this study. Moreover, Arps included all types of speech defects, and his tasks of motor efficiency were athletic feats requiring activity of the large muscles instead of fine motor action. Borovikov [8] reports that children with speech defects have poorer motor endowment, according to the Oseretzky scale for motor ability, than normal children.

Stinchfield and Young,[9] on the other hand, reported that in a study of 15 young children with delayed speech a better showing was made in performance tests than in the Binet or Kuhlman tests for mental ability. They based this conclusion on data for two tests. The high performance of their group on the Goodenough drawing test may well be comparable to the exceptional performance on the manikin test by the children in the present study. The second performance test was the Seguin formboard. Stinchfield and Young reported that a median performance of one hundred and ten seconds by their group with a median age of 6 years was an excellent accomplishment. These data, however, are open to another interpretation. According to the Pintner-Patterson norms and the Arthur norms, such a level of performance on the Seguin board is exceedingly poor. Indeed, it is even poorer than the performance shown on the Seguin by the experimental group of the present study. The data on Stinchfield and Young's group really serve to confirm the findings of the present study and lend weight to the conclusion that children with delayed speech seem to show retardation in their ability to do tasks requiring motor speed.

AUDIOMETRIC TESTS

The 2-A audiometric test was administered by an experienced tester. All tests except those of 3 controls were given at the New York League for the Hard of Hearing. When the results were at all doubtful, a retest was given. Only data for those patients in the experimental and control groups whose audiograms were believed to be accurate were retained.

Percentages of hearing loss for the right and for the left ear of each experimental subject and each control were computed according to an adaptation [10] of the Western Electric method.[11] The percentage of hear-

8. Borovikov, I. V.: An Investigation of the Motor Endowment of Children with Speech Disturbance (Logopaths) and of Deaf Mutes (Ocupaths), Voprosy izucheniya i vospitaniya lichnosti, 1926, nos. 2-3, p. 175.

9. Stinchfield, S. M., and Young, E. H.: Children with Delayed or Defective Speech, Stanford University, Calif., Stanford University Press, 1938.

10. Frequencies 256 and 4192 were given equal weight with 512, 1024 and 2048.

11. West, R.; Kennedy, L., and Carr, A.: The Rehabilitation of Speech, New York, Harper & Bros., 1937, p. 396.

ing loss for the individual members of the groups are shown in table 4. The median hearing loss for the right ear for the experimental subjects is 18 per cent, and that for the controls is 5.6 per cent; the median hearing loss for the left ear for the experimental subjects is 19.6 per cent, and that for the controls is 6 per cent.

The audiograms show the comparison between the median of each group.

An examination of figure 3 shows a superiority in hearing, in the entire range of frequencies, of the control group over the experimental. The discrepancy is especially striking in the highest frequencies. It will be noted that the child ranking lowest in speech has the most severe degree of hearing loss.

TABLE 4.—*Comparison of the Hearing Loss of Experimental Subjects and of Controls Judged by an Adaptation of the Western Electric Method*

Subject	Sex	Experimental Group: Percentage Loss		Control Group: Percentage Loss	
		Right Ear	Left Ear	Right Ear	Left Ear
1	♂	13.6	9.6	9.6	4.8
2	♂	20.8	19.2	7.2	7.2
3	♀	22.4	15.2	1.6	3.2
4	♀	4.8	20.8	3.2	2.4
5	♀	29.6	20.0	15.2	17.6
6	♂	33.6	36.0	4.0	11.2
Composite median.....		18.0	19.6	5.6	6.0

OBSERVATIONS

In the speech tests, the children with distorted speech were able to imitate isolated speech sounds surprisingly well, although all of the children were unable to combine them into words. They also made poor ratings in tests of articulatory speed.

The roentgenograms and the studies of the blood cholesterol did not reveal any significant difference between the two groups. Several children of the experimental group, however, in contrast to the control children, showed interesting physical variations which may be related to their distorted speech. One child showed a pronounced weakness of the palatal reflex. Another child manifested obesity possibly pituitary in origin. A third child had a turret-shaped skull, and a fourth child showed marked delay in the development of the centers of ossification, with no other clinical findings of hypothyroidism.

In the psychologic study the experimental group rated in the lowest level of the normal range. Only 1 child had an intelligence quotient as high as 107. At first we were inclined to regard this as partially due

to the low socioeconomic status of the patients. We soon discovered that this was not true, for in selecting our controls from the same clinic we found that we had to eliminate 3 or 4 controls for each experimental subject because of the higher intelligence quotient. In addition, the

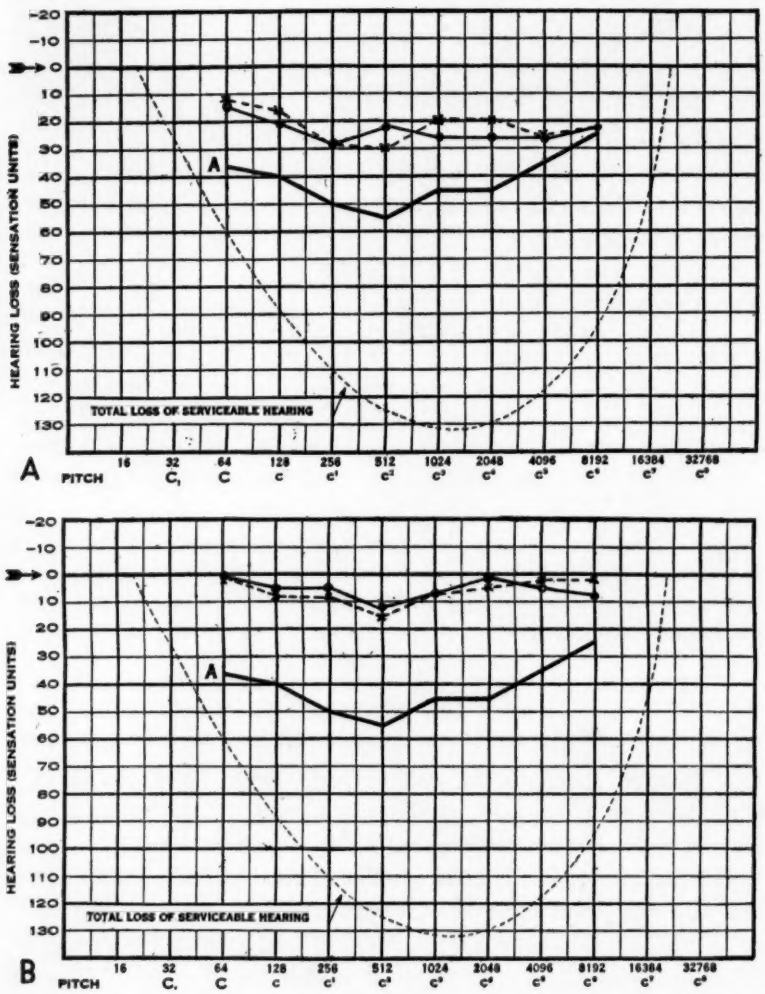

Fig. 3.—Audiograms showing the median hearing loss (*A*) of the experimental group and (*B*) of the control group. The arrow pointing to 0 in the horizontal column indicates normal hearing. The line at "A" indicates reading of audiometer when testing a person of normal hearing with the D-80904 bone conduction receiver. The per cent hearing loss for speech is approximately equal to the average reading at 512, 1024, 2048 multiplied by 0.8.

experimental group showed poor results in motor tests requiring speed. The results were especially striking in view of the fact that the tests did not demand any linguistic ability. This finding, if confirmed for larger

groups, is especially significant when it is recognized that speech requires rapidly shifting and delicately coordinated action of the various muscle groups and structures.

When we analyzed the audiometric tests, we found that the children in the experimental group, although many of them had diminished hearing, still had sufficient hearing ability to develop speech. As a group, however, the median hearing curve for the experimental subjects was lower than that for the controls, and the greatest difference appeared in the high frequency range.

On the basis of these findings, there are several possible explanations of the problem of distorted speech in young children.

We have studied these children with regard to their growth and development. While the terms growth and development are used interchangeably, growth refers to an increase in size whereas development implies an increase in function or complexity. Usually these two processes proceed simultaneously, having alternating periods of rapid and languid progress. Sometimes, however, growth may proceed at a normal rate while development may be somewhat retarded, or there may be considerable development and little growth. When this retardation of either development or growth occurs, it may be general or may manifest itself prominently in some special organ or system. Distorted speech may be regarded as an index of developmental retardation or deficiency in the speech areas, possibly associated with an accompanying deficiency in hearing acuity and motor coordination.

Another possibility is that the speech areas are normal and that the distorted speech is caused by the lessened acuity of hearing or the poor motor coordination or possibly both. Whereas it is known that profound congenital deafness prevents development of speech and that deafness acquired during early childhood causes regression in speech, investigators have not found a consistent relation between a lesser degree of hearing loss and functional articulatory disorders of speech. Since, however, the young children with speech disorders in the present series and also those described by Stinchfield and Young [9] present fairly marked diminution of hearing, we submit the theory that the presence of this hearing deficiency in the extremely young may be an important factor in the retardation of speech. In brief, the age at which the child becomes deficient in hearing may be the deciding factor as to whether or not deficient hearing tends to cause defective speech.

One also has to recognize the importance of psychologic factors in the problem of speech. The environment of the child must be a stimulating one. It must provide experiences out of which speech will grow, and it must present correct speech patterns for the child to imitate. Both these factors are vital forces in the motivation for normal development

of speech in the young child. Maladjustments at home, emotional instability in the parents or the child, negativistic tendencies or fear reactions may retard the normal development of speech.

Finally, endocrine factors, which play such an important role in the growth and development of the child, may have a determining influence in the etiology of distorted speech.

Some children who are exceedingly late in speaking—sometimes beginning as late as the age of 5 years—may spontaneously begin to speak and thereafter proceed at a somewhat accelerated rate so that they "catch up" with normal children. Case 2 gives an example of this spontaneous late onset of speech. We have also seen a pair of twins, girls, who at the age of 7 years used a type of speech so mutilated as to resemble invented speech (idioglossia) but who later, with no special speech help, had normal articulation.

However, we have also found at the college level many students, especially men, who retained the mutilated type of articulation. Such adults find progress in speech slow and laborious.

As to treatment, it is inefficient and uneconomical to postpone remedial measures in hope that the defect will be outgrown. In treating these patients the clinician uses many approaches. If he finds a condition calling for medical treatment, that condition should be the first to receive attention. Case 1 furnishes an example of this: The patient shows signs of physiologic retardation and is therefore being given thyroid. In addition to medical attention, the environmental factors, including home regimen, parental attitudes, school adaptation and emotional adjustments, should be appraised and improved in every possible manner. A specific speech program should be instituted, consisting of well motivated exercises in articulation in which appeal is made to every sensory avenue, the auditory, the kinesthetic and the visual.

SUMMARY AND CONCLUSIONS

In order to evaluate the factors associated with mutilated speech, 9 children who presented speech defect with no obvious physical cause were studied. Six of these were matched with an equally large group having normal speech. All the children were given physical, psychologic and audiometric tests. Three of the 9 subjects with mutilated speech showed delay in the centers of ossification, and 2 of the 6 controls showed delay. In a test measuring speed reproduction in speech, the children of the experimental group were extremely low, the highest reaching the sixth percentile.

Compared with the control group, the children with mutilated speech showed retardation in their ability to do tasks requiring motor speed. The experimental group was superior to the control group in tasks

requiring the organization of visually attractive materials when given unlimited time.

The control group showed a median hearing loss of 5.8 per cent, whereas the experimental group showed an 18.8 per cent loss. The greatest difference between the two groups appeared at the higher frequencies.

In conclusion, it appears that even in this relatively homogeneous group a number of factors are associated with dyslalia. These factors, as exhibited by our group, are: hearing loss, especially in the higher frequencies; a lack of motor speed; endocrine dysfunction, and unfavorable environmental relation. These factors, singly or in combination, may operate to produce dyslalia.

The cases of the 3 patients who were excluded from the experimental group will be reported now.

REPORTS OF THREE CASES

CASE 1.—S. Y., a boy aged 6 years 4 months (Vineland score 81, with a social age of 4 years 10 months; Binet score, 64; score for performance cubes test, 65; Healy score, 97; Seguin score, 75; score for mare and foal test, 112, and manikin score 65), was a "behavior problem" child who had been barred from the public school until such time as he should learn to speak. He was a disrupting influence at the speech clinic but gradually learned to cooperate in the group work, at least to the extent that it was possible to permit him to remain.

The physical examination gave negative results. A roentgenogram of the wrists revealed a slight delay in development of the centers of ossification.

His speech was practically incomprehensible. In addition to the fact that many of his sound units were lacking and others faulty and the fact that he used elisions in the pronunciation of words, he used many Yiddish words while attempting to speak English.

The audiometric test was first attempted at the New York League. The tester reported that she had not been able to secure the subject's cooperation but was reasonably sure that there was a hearing loss. One of us made two attempts to get an accurate audiogram of this subject but failed, owing to the great distractibility and suggestibility of the boy. She, too, felt certain that there was a hearing loss.

The subject had a feeble-minded older brother, who had defective speech. His mother was probably of low mentality also, since she conducted herself like a restless child and in conversation revealed a dearth of ideas. She used broken English scarcely more comprehensible than her son's language. It was believed that the speech status could be explained fully on the basis of: (1) lack of good speech model, (2) lack of discipline and habits of attention, (3) hearing loss, (4) lack of environmental stimulation and (5) inferior mentality.

CASE 2.—E. T. a girl of 3 years 6 months (intelligence quotient 83; Seguin score, 98?, Vineland score, 121, social age of 4 years 3 months) understood what was spoken but used practically no speech and would make no attempt to do so. Her vocabulary consisted of one or two words, "mama" and "Arthur," the name of her brother. It was difficult to get her to cooperate in the speech test; in fact she did so only when her mother bribed her. The bribe consisted of a quarter, the child refusing to respond to the promise of an ice-cream cone or to the offer of a nickel.

With a quarter in sight on a chair beside her, the little girl made all the articulatory movements except that for "l" and imitated the experimenter's vowels, diphthongs and consonants except "sh," "h," "p-b," "f-v," "ng," "r," "s" and "l".

In the course of the psychologic examination the mother continually corrected the child when she attempted to handle the test materials with her left hand instead of the right. E.T. seemed to resent this, and the examiner suggested that the mother allow the child to use the preferential hand. Because of the child's extreme negativism, possibly owing to the constant nagging about the use of the left hand, the examiner could gain her cooperation only on the nonverbal items, and the prorated quotient of 83 which resulted is probably too low. On the Vineland social maturity scale the mother's social quotient was 121.

When the child was seen six months later, she had greatly improved in behavior and in speech. She responded about as would any other child of her age and used speech appropriate for a 4 year old. The mother reported that she no longer corrected the child when she used the left hand. The improvement in speech may have resulted from the improved emotional relation between mother and child.

CASE 3.—A. R. is a shy boy of 4 years 7 months, dominated by neurotic parents. The father and an older brother are stutterers. The father was a markedly neurotic person who attributed his economic failure to his speech defect. He was very exacting with A. During the psychologic examination the father at first pounced on the boy and shook him whenever he gave the wrong answer. When the examiner refused to permit this, the father paced up and down at the far end of the room wringing his hands and beating on the wall with clenched fists. A.R. seemed to ignore this disturbance and cooperated well, earning the following score: Binet 89, Vineland 91, and a somewhat erratic performance on the motor tests.

The results of the physical and laboratory examinations were entirely normal.

A later interview with the mother showed her to be highly emotional also. The child appeared to be afraid of his parents, yet entirely dependent on them. The unwholesome emotional tension in the home instilled into the child a sense of fear and insecurity and may have played a large role in his speech retardation.

EFFECT OF IRRADIATION OF THE AIR IN A WARD ON THE INCIDENCE OF INFECTIONS OF THE RESPIRATORY TRACT

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Acute infections of the upper part of the respiratory tract offer at the present time the most important medical problem for those who are caring for young infants in institutions as well as in pediatric wards of hospitals. Diarrheal diseases have been controlled in institutions and throughout the community. The mortality, which used to be highest in the summer, has now shifted to the winter and early spring months, the period of prevalence of infections of the upper part of the respiratory tract which are characterized by many complications including, besides pneumonia, otitis media, mastoiditis and pyuria. There is no doubt that the morbidity due to infections of the upper part of the respiratory tract in institutions housing infants has been not only great but appalling. An infection of this kind occurring in a ward acts like a contagious disease, reaching at times epidemic proportions. It is for this group of diseases, in which the causative agent enters and leaves through the nasopharynx, that no practical means of control has yet been evolved.

We shall review briefly some of the prophylactic measures attempted during the past thirteen years at the Home for Hebrew Infants. In 1925 we made use of the mercury vapor lamp, exposing a group of 21 infants to the rays for a period of four winter months.[1] The treatment did not lessen the number of infections in this group as compared with the control group. Since the rays from the carbon arc lamp more nearly approach the spectrum of the sun, we tried the effect of this lamp in reducing infections during 1926.[2] A controlled group of 9 infants was systematically exposed for a period of three winter months. This form

From the Home for Hebrew Infants.

1. Barenberg, L. H.; Friedman, I., and Greene, D.: The Effect of Ultraviolet Irradiation on the Health of a Group of Infants, J. A. M. A. **87**:1114 (Oct. 2) 1926.

2. Barenberg, L. H., and Lewis, J. M.: The Effect of Carbon Arc Irradiation on the Health of a Group of Infants, J. A. M. A. **90**:504 (Feb. 18) 1928.

of prophylaxis also failed to cause a diminution in the incidence of respiratory infections. In 1928 our next attempt was to determine whether the spread of infections during the winter could be prevented by mechanical means, by breaking the bond of contact through "aseptic nursing."[3] The results of this investigation were unsatisfactory.

It was evident that we could not depend on ultraviolet rays nor on "aseptic nursing" as a measure for decreasing the incidence and the spread of respiratory infections. All attempts at creating artificial immunity were then discontinued, and we next turned our attention to nutrition. It was thought that a review of our experiences of the previous twelve years might afford information of value for conducting a campaign against disease of the respiratory tract. A careful survey, in 1929,[4] of our infant population showed that nutrition, as gaged mainly by body weight, does not bear any relationship to the occurrence of pneumonia or to the frequency with which infection of the respiratory tract develops in children. In 1930 a clinical study was undertaken to compare the incidence and severity of infections of the respiratory tract among four groups of infants receiving various quantities of vitamin A added to their diets.[5] The results of this investigation showed no significant differences in the four groups; the one which received the largest amount of vitamin A daily was not protected against infection to any greater extent than the other groups.

Renewed interest in the prophylactic treatment of cross infections was stimulated by the researches of Wells and Wells,[6] in which a new type of instrument and a new technic were used for the study of the dispersion of bacteria in the air and the effect of physical and chemical agents on their viability. These investigators noted in the air of enclosed spaces dried residues of infected droplets derived directly from droplets less than 0.1 mm. in diameter. These droplet nuclei floating in the air are peculiarly adapted to bear infected matter. When a person sneezes or coughs, many thousand nasopharyngeal organisms remain suspended in the air, and in occupied enclosed spaces exchange of nasopharyngeal flora is inevitable. They also found that radiations from ultraviolet lamps are efficient in destroying micro-organisms suspended in the air of enclosed spaces.

3. Abramson, H., and Barenberg, L. H.: Respiratory Disorders in Infants: Attempted Prevention by Control of Contact Infection, J. A. M. A. **92**:2156 (June 29) 1929.

4. Barenberg, L. H.; Greene, D., and Abramson, H.: The Relationship of Nutrition to Pneumonia in Infancy and Childhood, J. A. M. A. **92**:440 (Feb. 9) 1929.

5. Barenberg, L. H., and Lewis, J. M.: Relationship of Vitamin A to Respiratory Infections in Infants, J. A. M. A. **98**:199 (Jan. 16) 1932.

6. Wells, W. F., and Wells, M. W.: Air-Borne Infection, J. A. M. A. **107**: 1698 (Nov. 21); 1805 (Nov. 28) 1936.

The practical application of the studies of Wells and Wells was made by Hart [7] in the operating room and by McKhann in a children's ward. Hart obtained favorable results in the reduction of the number of traumatic infections, and he found that the effectiveness of ultraviolet radiations depends on: (1) the type of organism, not all organisms being equally susceptible to the rays; (2) the distance of organisms from the lamp; (3) the velocity of the passage of the organisms through the rays, and (4) the character and source of the radiations. McKhann [8] attempted to eliminate the transmission of the chickenpox virus through the air by a barrier of ultraviolet radiation. Twelve patients with chickenpox were kept in the ward throughout the duration of their illness. For a period of five months the children in this section were separated from the rest of the patients by a barrier of ultraviolet radiation. Only 1 child of 120 beyond the barrier acquired chickenpox.

One of the most striking examples of an outbreak of respiratory infections, presumably air borne, was reported by Sauer and McDonald.[9] An outbreak was carefully traced in an aseptically conducted nursery, the Cradle, in Evanston, Ill., in which 10 of 11 infants and 6 members of the medical and nursing staff were attacked by infections of the respiratory tract.

The present investigation was undertaken primarily to compare the incidence and severity of infections of the upper part of the respiratory tract among a group of infants living in a ward where the air was irradiated by ultraviolet rays and those among a similar group of infants housed in a ward where the air was not irradiated. Because of our limitations, bacteriologic studies of the flora of the air and of the noses and throats of the infants could not be carried out. This investigation was, therefore, purely clinical. This study covered two periods, one of four and a half months, from Jan. 5, 1938 to May 31, 1938, and another of six months, from Oct. 14, 1938 to April 18, 1939, seasons when the incidence of infections of the upper part of the respiratory tract is at its peak.

METHOD

For the purpose of this study we selected two wards identical in their construction but situated on different floors. These wards measure 37 by 25½ by 10 feet (11.3 by 7.8 by 3 meters) and have a volume of 9,435 cubic feet (268

7. Hart, D.: Sterilization of the Air in the Operating Room by Special Bactericidal Radiant Energy: Result of Its Use in Extrapleural Thoracoplasties, J. Thoracic Surg. **6**:45 (Oct.) 1936. Hart, D., and Gardner, C. E., Jr.: Sterilization of the Air in the Operative Region with Bactericidal Radiant Energy, Tr. South. S. A. **49**:377, 1937.

8. McKhann, C. F.; Steeger, A., and Long, A. P.: Hospital Infections, Am. J. Dis. Child. **55**:579 (March) 1938.

9. Sauer, L. W., and McDonald, J. J.: A Midsummer Respiratory Infection in an Aseptically Conducted Adoption Nursery, J. Pediat. **14**:304 (March) 1939.

cubic meters). Since we decided not to regulate the flow of air through these rooms, the windows were either opened or shut, according to the room temperature and the temperature outside. The cribs were evenly spaced about the room and separated from one another by a distance of 2 feet (0.6 meter). In the center of the room were placed "Jimmie Jumpers," in which the infants would sit and exercise for variable periods during the day.

Two ultraviolet lamps were installed in one of the wards diagonally opposite each other at a distance of 20 feet (6 meters) and 12½ feet (3.8 meters) from the respective corner, as shown in the accompanying photograph. The floor type of the Safe-T-Aire ultraviolet lamp, which is above the eye level, was used throughout the study. It is a quartz mercury vapor lamp emitting rays of wavelengths between 2,500 and 2,600 angstrom units, and operates directly from 100

The ward where the air was irradiated by ultraviolet rays.

to 120 volt, 60 cycle alternating current. According to this arrangement of the lamps, the infants while they were in their cribs were at a distance ranging from 6½ to 19 feet (2 to 5.8 meters) from either one of the lamps, and when they were in the "Jimmie Jumpers," were at a distance of 10 feet (3 meters) from the lamp. The schedule of operation of the lamps was such that they were turned on for half an hour and off for half an hour from 6 a. m. to 8 p. m. daily—a total irradiation of seven hours per day.

It will be of advantage to consider briefly the age group selected for our study. Experience has taught us that by the time a child reaches the age of 2½ to 3 years he is less susceptible to infections of the respiratory tract than a younger child; at any rate, complications are less apt to develop. Moreover, controlling a group of older children for a study of this kind would present a difficult problem. Similarly, infants under 4 months of age still retain the immunity which they

obtained from the mother. There is great danger of error in translating the results of observations of young infants into terms of older children. We therefore selected infants between 7 and 16 months of age, a period when the original immunity has been lost and immunity has not yet been acquired through repeated infections.

The infants were divided into approximately an equal number between the control and the irradiated ward. Admission to the wards was discontinued until the end of the study. Only when an infant contracted an infection was he removed to the hospital building, and he was brought back to the ward on the termination of the infection. The children were visited weekly by their parents for one-half hour at each visit. All infants received a diet adequate in every respect. Complete monthly physical examinations were made on each infant.

The personnel of each ward consisted of two permanent and two transient nurses, the latter being in the ward at least two months.

Temperatures of the infants were taken routinely twice a day; when an elevation of the temperature was present, the reading was taken every four hours.

The classification of infections was based on the presence of fever with certain signs and symptoms. The infections were grouped into four types: (1) those with a temperature of 100 to 101 F. for one or two days, accompanied by a slight rhinitis, were designated as very mild; (2) those with a temperature of 101 to 103 F. for two or three days, associated with a congested pharynx, moderate rhinitis and anorexia, were considered mild; (3) infections were labeled moderate when the temperature rose above 101 F. for three days or longer and was accompanied by marked congestion of the pharynx or tonsillitis, prostration and anorexia; (4) pneumonia, purulent otitis media, septic sore throat and toxemia were designated as severe infections.

RESULTS

A glance at tables 1 and 2 shows that during the first period of study, from January to May 1938, there was a marked diminution both in the total number and in the percentage of moderate and severe infections in the irradiated ward as compared with the control ward. Only 37 infections occurred in the irradiated ward, while in the control ward 73 infections developed. It is of interest to note that only 3 infections which were classified as moderate and severe occurred in the experimental ward, whereas 14 infections of these types developed in the control ward. This is brought out more definitely by the last column in table 1, in which it is shown that during the period of observation the number of infections per child per month in the irradiated ward amounted to 0.42 while in the control ward the number of infections per child per month was 0.77. In other words, there were approximately twice the number of infections in the control ward as in the irradiated ward. A more marked difference occurred between the two wards with regard to the number of moderate and severe infections; namely, the number of moderate and severe infections in the control ward was about five times that of the irradiated ward. As revealed by the values of "P" in the last column of table 2, there is a real significance in the difference between the number of total infections in the irradiated

ward and that in the control ward. This value of "P" indicates that the differential in the relative frequency of infections between the two groups of infants is much greater than can be ascribed to chance alone.

TABLE 1.—*Comparison of the Incidence and Severity of Infection of the Upper Part of the Respiratory Tract Between Two Groups of Children During Four Winter Seasons (1935-1939)*

Date	Ward	Number of Children	Average Age at Beginning of Study, Mo.	Average Period of Observation, Mo.	Infection						
					Total Number	Very Mild	Mild	Moderate	Severe	Percentage of Moderate and Severe	Per Child per Month
10/14/35 to 4/18/36	1	22	10.4	4.7	60	41	12	3	4	11.6	0.57
10/14/35 to 4/18/36	5	21	11.6	4.0	43	31	5	7	0	16.3	0.51
10/14/36 to 4/18/37	1	18	10.4	4.6	58	43	9	5	1	10.3	0.7
10/14/36 to 4/18/37	5	19	12.0	4.6	56	43	8	5	0	9.0	0.64
1/ 5/38 to 5/31/38	1*	18	8.5	4.87	37	24	10	2	1	8.1	0.42
1/ 5/38 to 5/31/38	5	19	8.1	4.9	73	45	14	9	5	19.1	0.77
10/14/38 to 4/18/39	1*	17	9.0	5.6	56	38	13	4	1	8.9	0.59
10/14/38 to 4/18/39	5	20	10.5	5.6	93	61	20	11	1	12.9	0.78

* Ward in which the air was irradiated by ultraviolet rays.

TABLE 2.—*Summary of Incidence and Severity of Infections in the Control and the Irradiated Ward*

Date	Infection	Control Ward	Irradiated Ward	Difference	"P"*
1/5/38 to 5/31/38....	Total number.....	73	37	36	<0.01
	Moderate and severe	14	3	11	0.01
10/14/38 to 4/18/39..	Total number.....	93	56	37	0.04
	Moderate and severe	12	5	7	0.17

* "P" is a measure of the extent to which the differential in the relative frequency of infections between the control and the experimental groups may occur by chance. When "P" is less than 0.05 the differential between the control and the experimental groups is much greater than can be ascribed to chance alone. The values of "P" shown in this table were derived by means of the chi square test (Hill, A. B.: Principles of Medical Statistics, London, The Lancet, Ltd., 1937).

Of similar significance is the differential of severe infections between the control and the irradiated ward.

During the second period of study, from October 1938 to April 1939, although the results were not so striking as those of the first period, nevertheless there was a significant difference in the total number of infections between the two wards. Computing the findings on the basis

of 20 infants per ward, we found that there was a total number of 66 infections in the irradiated ward as compared with 93 infections in the control ward. The number of infections per child per month in the irradiated ward was 0.59, whereas in the control ward it was 0.78. Statistical analysis of the results for this period shows that there is a real significance in the value of "P" for the total number of infections, but the value of "P" for the severe infections shows this difference not to be significant.

Since the wards under consideration always have had a group of infants similar to that of the present study, an attempt was made to compare the frequency and severity of infections in these two wards during the two years preceding the present study, at the same seasons of the year. As table 1 reveals, ward 1, which is the irradiated ward in the present study, had practically the same number of infections per child per month as did ward 5, the control ward of the present study, during the winter and early spring months of the year 1935-1936. Similarly, an equal number of infections occurred in these two wards during the winter season of 1936-1937. There was also no significant difference in the severity of infections in these wards during these two seasons. Obviously two similar groups of infants living under the identical environmental conditions can be expected to have a similar incidence of infection when no prophylactic measures are carried out to diminish the number of infections in one of the groups.

COMMENT

It is realized that a study of this kind should be carried out for a number of years as the type and severity of infection are subject to seasonal variations, and it is therefore premature to lay down rigid conclusions as a result of this study. Moreover, the form of therapy under consideration in this paper is directed primarily to the source of infections of the respiratory tract which are brought about by the inhaling of infectious material carried in the air. Therefore, the clinical observations should be fortified by accurate studies of the flora of the nose and throat of each infant as well as by studies of the bacterial content of the air before and after irradiation. However, after an observation on a well controlled group of children over a period including two winter seasons, supplemented by a survey of the infections that occurred during the two previous winter seasons in the same ward, we may state from a purely clinical point of view that this form of prophylaxis seems to offer greater promise than the other methods previously employed. It has had greater effect in inhibiting infections of the respiratory tract and in reducing the number of their complications to some extent.

According to the routine carried out in our study, the air in the ward was being irradiated intermittently from 6 a. m. to 8 p. m., one-half hour on and one-half hour off; in other words, seventeen hours out of twenty-four the air in the ward received no ultraviolet rays. Wells and Wells [6] have demonstrated that bacteria entering the air from the nasopharynx as a result of sneezing and coughing do not die immediately but remain in the air long enough to be transmitted to other persons. We believe, therefore, that a further reduction in the incidence of infections of the upper part of the respiratory tract may be accomplished by more intensive and prolonged treatment. We are planning to carry out this form of therapy next winter in the form of a continuous instead of an interrupted sterilization of air.

SUMMARY

The failure of previous attempts at prophylaxis to reduce the incidence and severity of infections of the upper part of the respiratory tract is discussed. The effect of the sterilization of air in a ward by means of ultraviolet radiation on the incidence and severity of infections of the upper part of the respiratory tract, as studied during two winter seasons, is reported.

A group of infants ranging in age from 7 to 16 months were placed in a ward where the air was irradiated intermittently, one-half hour on and one-half hour off from 6 a. m. to 8 p. m. daily for a period of four and a half months in 1938. A similar group of children were observed during the same period in a ward where the air was not irradiated. Similar observations were carried out for a period of six months in 1938-1939. A definite decrease in incidence and in severity of infections of the upper part of the respiratory tract was noted among the children who were kept in the irradiated ward as compared with those in the control ward during 1938. In the subsequent study, though the reduction was not as striking as in that of 1938, nevertheless a significant reduction in the total incidence and to a slight extent in the severity of infections of the upper part of the respiratory tract was obtained among the children in the irradiated ward as compared with those in the control ward. The possibility of further reduction in the incidence and severity of infections of the respiratory tract by means of continuous irradiation is suggested.

1749 Grand Concourse.

910 Grand Concourse.

1560 Grand Concourse.

Mr. M. Spiegelman, of the statistical bureau of the Metropolitan Life Insurance Company, gave assistance in the statistical complications.

VIII. BACKGROUND AND SOCIAL ADJUSTMENT OF THYROID-DEFICIENT CHILDREN RECEIVING GLANDULAR THERAPY

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Many studies have been reported concerning the effect of medical treatment on the physical development of thyroid-deficient children, and several concerning the mental growth of such children, but few dealing with their social adjustment. In a former paper the question was raised as to the value of thyroid therapy in the ultimate adjustment of these children, as they tend to remain at a relatively low level of mental development.[1] The purpose of this study is to report the background and social adjustment of 27 children deficient in thyroid who have received medication over a period of seven years.

MATERIAL

The material for this study was gathered from information secured during visits to the homes and the schools, from interviews with the parents and the patients and from the records of social agencies which have been in contact with the families.

The close relationship between the pediatrician and the parents insures to a degree the reliability of the material. In most instances the parents were willing to cooperate in providing the necessary information, as they were satisfied with the progress the children had shown under thyroid treatment.

Cultural and Economic Background.—The distribution of the nationality of the parents is as follows: Twenty-seven were born in the United States; 9 in Russia; 3 each in Poland, Norway, Austria and

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Based on a thesis presented to the Smith College School for Social Work in August, 1936.

From the Institute for Juvenile Research and the Department of Pediatrics, University of Illinois College of Medicine and the Research and Educational Hospital.

1. Bronstein, I. P., and Brown, A. W.: Hypothyroidism and Cretinism in Childhood: II. Mental Development, Am. J. Orthopsychiat. **4**:413 (July) 1934.

Czechoslovakia, and 1 each in Germany, Ireland, Scotland, Italy, England and Lithuania. It is interesting to note that none of the patients was Negro.

The parents of these children seem to have come from fairly large families. In 23 of the cases the average number of children in the father's family is 5, with a range of 3 to 14, while in 22 of the cases the average number of children in the mother's family is 5, ranging from 2 to 16. On the other hand, the patients themselves come from fairly small families, the average number of children being 2, with a range from 1 to 9.

The formal education of the parents is about that which would be found in any random selection of the population. Nearly all the parents have less than a four year high school education, but the range of schooling is from four grades of school to four years of college. Nothing of significance appears in connection with the occupations of the parents. They are distributed among the unskilled, semiskilled, skilled and professional groups. Twenty-one families are self supporting; 6 families are being assisted by relatives or the public relief agency, the fathers being unemployed. Nineteen of the 27 families have been known to one social agency or more.

There is a large range in the type of home and neighborhood from which these patients come. The majority live in lower middle class neighborhoods in crowded quarters; 6 live in poor foreign neighborhoods, while 2 live in large homes in good residential districts. Eight of the homes were not visited, as the families live some distance from the city.

Subjects.—For purposes of comparison the patients are divided arbitrarily into four age groups: group I, under 7 years of age; group II, from 7 through 12 years of age; group III, from 13 through 16 years, and group IV, from 17 through 21 years.

GROUP I: CHILDREN ONE THROUGH SIX YEARS OF AGE

This group consists of 4 children. The following tabulation gives the sex, age and level of intelligence of each child.

Serial Number of Patient	Sex	Age	I. Q.*
1	F	15 mo.	Dull or borderline
2	M	2½ yr.	Dull
3	F	3½ yr.	Dull
4	F	4 yr.	Imbecile

* Ratings on children of this age group are known to be unreliable. In this study the following classification of intelligence quotients was used: 0-19, idiot; 20-44, imbecile; 45-69, high grade defective; 70-79, borderline defective; 80-89, dull; 90-109, average.

At the time treatment was instituted these children had the typical characteristics of the hypothyroid child. Three have made remarkable

physical improvement and at present cannot readily be recognized as having a thyroid deficiency or as being mentally retarded. Child 4 has shown some physical improvement, but still looks and acts like a mentally defective child. Marked changes in personality in all of the children followed treatment. Before therapy they appeared dull, sluggish and little interested in their surroundings; after therapy they became more alert, active and interested in their environment. Before coming to the hospital many presented feeding problems, but since they have been receiving the treatment their appetites have become good. Their play interests seem to be similar to those of normal children of the same mental age.

The following case report is fairly typical for children in group I who have responded favorably to treatment and thus far have made a fair social adjustment:

PATIENT 3.—The patient is a 3½ year old girl, the younger of two siblings of American-born parents in whose family there is no history of a thyroid disorder. The father had an eighth grade education and was a mechanic by trade; by a previous marriage he had 4 normal children. The mother, who was fourteen years his junior, is young, attractive and intelligent but emotionally immature; she is one of 8 children. She described her marriage as "jumping from the frying pan into the fire," for she became pregnant immediately and had difficulty with the step children, and shortly afterward the father began to drink to excess. There is a long history of marital discord, with frequent visits to the court of domestic relations and with frequent separations, during which time eight social agencies had contact with the family. The father was unemployed for five years, and the family received financial assistance from the public relief agency during that period. When the patient was 3 years old the father committed suicide while intoxicated. The mother appeared little disturbed by his death and has continued to maintain a home for her 2 children and the stepdaughter in crowded quarters in a lower middle class neighborhood. The patient's 6 year old sister, who is in the first grade, is a nervous child and presents a feeding problem.

The mother was 28 and the father 42 at the time of the patient's birth. The child was wanted, but the father abused the mother during the pregnancy. The birth was normal, the patient weighing 6¾ pounds (3,061.74 Gm.). The mother reported that the child stopped developing at less than 3 months of age, when breast feeding was discontinued. Soon after this the infant "croaked like a frog," her abdomen distended, and she frequently strangled. She slept poorly and became constipated.

The mother made many unsuccessful attempts to secure help for the patient, finally bringing her to the clinic when she was 3 months old. She weighed less than 7 pounds (3,175.14 Gm.), and had a large tongue, an umbilical hernia and a pudgy face with dry yellow skin. The extremities were short in proportion to the trunk. Treatment with thyroid was begun immediately, and within a month there was a "miraculous change." Dentition began when she was 5 months old, and toilet habits were established when she was 1½ years old; she began walking at 2 years of age. An intelligence test when she was 12 months old classified her as dull.

The patient is now an attractive looking 3½ year old brunette child, 39½ inches (100 cm.) tall and weighing 33¼ pounds (15 Kg.). She is well nourished and

unusually active. Her coordination is poor; she talks rapidly and has infantile speech. It was impossible to give her a rating on the last psychologic examination, but it was judged that she could still be ranked in the dull group. She dresses herself, cares for her bodily needs, eats well and has good table manners. She likes music, enjoys singing and has performed before large groups with no hesitation. She gets along best with younger children, usually taking a passive role. The mother has difficulty in disciplining her and finds that the child is very tiring. The mother seems to favor her, but apparently there is little conflict with the sister over this. The mother is hopeful about the patient's future and has accepted her mental retardation since she has become so attractive physically.

The patient of the following case report is an example of children who have not responded to the treatment and have made a poor social adjustment:

PATIENT 4.—The patient is a 4 year old girl, the younger of 2 siblings. The father is a borderline mental defective; he is unkempt and habitually unemployed and has been extremely cruel to his family. The mother was born in Lithuania and came to this country when a small child. The maternal grandmother was always considered peculiar, but there was no history of a thyroid disorder in the family of either parent. The marriage was an extremely unhappy one because of the father's brutality, and the children were badly neglected. The father deserted the family; later, on three occasions, the mother was committed to a state mental hospital, finally with a diagnosis of dementia praecox of the catatonic type, with mental deficiency. Eleven social agencies have known the family, and finally one of these accepted responsibility for the patient. Her 10 year old sister, who is doing good work in the fifth grade, has made a relatively good adjustment in a foster home.

Little is known of the patient's developmental history except that adoption was discouraged because hypothyroidism was immediately suspected. When she was admitted to the hospital at the age of 13 months she was 32¾ inches (83 cm.) tall and weighed 22 pounds (10 Kg.). She had coarse, scaly skin, a large tongue, a protruding abdomen and a hoarse cry. She was unable to sit or hold up her head, and it was necessary to feed her. A psychologic test at this time placed her in the imbecile group. After some glandular treatment she showed marked physical improvement; her skin softened, dentition was accelerated, and her facial expression changed; but she did not respond either mentally or socially. She made a poor adjustment in a foster home and was finally placed in an orphanage.

At 4 years of age the patient is a tall, slender, unattractive blond child, obviously a low grade mental defective. She is 37 inches (94 cm.) tall and weighs 34 pounds (15.4 Kg.). She has a peculiar gait, and does not talk. She has incontinence of urine and feces. She can dress herself and will amuse herself for hours with any toy. She has an enormous appetite and is like a "mad person" if food is withheld. She has violent fits of temper, is incorrigible and is so rough with other children that isolation is imperative. Plans are being made for her commitment to a state institution for the feebleminded.

Comment on Group I.—Three of the 4 children of this age group are accepted by their parents and are making a satisfactory social adjustment within a limited environment. The difficulty of patient 4 in making an adequate adjustment seems to be related to her very low intelligence rather than her hypothyroid condition; the low mentality in turn may be

due to an unstable constitution, since her mother is a psychotic mental defective and her father was a borderline mental defective. Moreover, she does not have the security within a family group which the other children have.

GROUP II: CHILDREN SEVEN THROUGH TWELVE YEARS OF AGE

There are 11 patients in this group. The following tabulation presents the sex, age, intelligence quotient and formal education of each one:

Serial Number of Patient	Sex	Age	I. Q.	Education
5	F	7	55	Kindergarten
6	M	8	63	1st grade
7	M	8½	74	2nd grade
8	F	8½	71	1st grade
9	F	9	72	1st grade
10	F	9	93	2A grade
11	F	9	33	No attendance at school
12	F	9	100	3B grade
13	M	9½	42	No attendance at school
14	F	10	61	3B grade
15	M	12	57	Special room

All but 3 of these children looked like typical cretins when they came to the clinic. Since receiving treatment all have made remarkable physical improvement and, with the exception of patient 15, cannot now be recognized as having thyroid deficiency. Patient 12 stutters, and patient 11 has difficulty in talking clearly.[2] Three of these children, patients 5, 6 and 7, have made a relatively good social adjustment; 4 children, patients 10, 13, 14 and 15, have made a fair adjustment; 4 children, patients 8, 9, 11 and 12 have adjusted poorly.

The 3 children who have made a relatively good adjustment eat well, have few sleep disturbances, engage in the usual children's games and get along well with their playmates. According to the parents, all are easily disciplined, and they seem to be accepted in their respective homes.

The patient in the following case report is an example of children in this age group who have made a relatively good social adjustment:

PATIENT 7.—The patient is an 8½ year old boy, an only child. The father is one of 7 children born in this country in a famliy in which there is no history of a thyroid disorder; he completed the eighth grade and then made a fair income doing odd jobs. The mother, the fifth of 8 children of German-born parents, finished the eighth grade and did stenographic work for seven years after being graduated from a business college. The parents now eke out a living by operating a tavern in the village where they live. They have never been known to any social agency.

The mother was 24 and the father 25 at the time of the pregnancy. The former was well during gestation, but the birth was difficult, a forceps being used in the delivery. The patient weighed 8½ pounds (3,855.5 Gm.) at birth. He was

2. Schreiber, S.; Bronstein, I. P., and Brown, A. W.: Speech Studies in Cretins, J. Nerv. & Ment. Dis., to be published.

breast fed for six months; dentition began when he was 1 year old and talking and walking when he was 2 years old. He had a thick tongue and a protruding abdomen. When he was 1 year old his condition was diagnosed as hypothyroid, and thyroid was administered for three years. The patient was 5 years old when he was admitted to the hospital. During the eleven months prior to admission he had no treatment, and some hypothyroid characteristics developed. He weighed 65 pounds (29.5 Kg.) and was 4 feet 3 inches (129.5 cm.) tall. A psychologic test at this time found him to have an intelligence quotient of 69. With renewal of the treatment he began to grow taller and gained in weight in spite of his poor appetite.

The patient is now an attractive 8½ year old brunet boy; he has a friendly manner and is always neatly dressed. An intelligence quotient of 74 was secured on his last mental test. He is usually a sound sleeper, though occasionally he screams. He has been enuretic for a long time. His appetite has improved greatly since he has liked playing out of doors. When younger he was not interested in play, but this interest has developed with the continuance of the treatment. He gets along well with the few children in his neighborhood and is no longer completely dominated by his close friend. He fantasies a great deal, which the mother attributes to the fact that he has few companions. He sings, and he learns music easily. He likes running games. He has a horror of animals, being afraid of both dogs and cats until he knows them. He is disciplined easily but has an occasional fit of temper. The patient expects to be passed to the third grade in the fall. The teacher is aware of his hypothyroid condition and attempts to help him with arithmetic, which he finds difficult; as a result, he likes both her and his studies. A recent achievement test showed that he has good reading mechanics, which seem to be in advance of his comprehension. The parents think that the treatment has been successful, and they apparently are able to accept his intellectual limitations.

The 4 children making a fair adjustment all eat well and dress themselves; only 1 has night terrors; 2 are enuretic. All have some difficulty in getting along with other children. Three are reported doing poor work in school. Two get along well with their siblings; the younger brother of 1 resents having to play with his retarded sibling. The mother of 1 patient seems to reject her daughter, perhaps because the child is unattractive owing to superfluous hair on her face. The mother of another of the patients professes acceptance of the condition, although she is very irritable with the child. The other children in this group seem to be accepted by their parents, though the mother of 1 is disappointed because her daughter is unable to help her with household duties.

Patients 8, 9, 11 and 12, who have made a relatively poor adjustment, eat well but have many more disturbances during sleep than do the other children. Patients 8 and 11 are known to masturbate, while patient 8 bites her nails, and patient 12 does not dress herself. These children are easily disciplined, except patient 11, who is quite incorrigible. They have play interests similar to those of other patients in group II, but all have difficulty in getting along with other children. Patient 11, who has the lowest intelligence quotient, has never attended

school; patient 9 has been in school for only six months; patient 8 is repeating the first grade; patient 12 is doing good work in 3 B grade but has difficulty in getting along with her classmates. Patient 8, who is jealous of her sisters, quarrels with them continually; patient 9 feels resentful toward her sister, and patient 12 dislikes her brother. The mother of patient 8 is overly solicitous, and the mother of patient 9 is disappointed in her child's lack of responsibility. Patient 11, though attractive physically, is rejected by her mother because of her low mentality and incorrigibility; the mother of patient 12 is overanxious, setting too high standards for her daughter with no appreciation of her present achievement. In each case the parents, except those of patient 9, who said that their child was better before the treatment because she was not so noisy and active, feel that their child has improved physically but has not shown comparable mental growth.

Patient 8 may be used as an example of children in this age group who have made a relatively poor social adjustment.

PATIENT 8.—The patient is an 8½ year old girl, the second of 4 siblings. Her father, the fourth of 5 children, was born in Scotland and came to this country at an early age; at present he works as a truck driver. There is no history of a thyroid disorder in his family, but he did not talk until he was 7 years old. Two maternal aunts and the maternal grandmother have goiters. The mother, who is of Polish parentage, received an eighth grade education, worked as a beautician and at 17 married against the wishes of her parents. All 4 children are in good health. The patient's 12 year old sister is in the sixth grade, a 5½ year old sister is in the first grade with the patient, and the youngest child is 2 years old. The family has financial difficulties but lives in a comfortable home in a lower middle class neighborhood; a medical clinic is the only social agency with which this family is registered.

The mother was 20 when she became pregnant with the patient after a fourth miscarriage. She says she wanted to have a baby but was anemic; since then a goiter has developed, which has never been treated. The birth was premature but normal. The mother was unable to nurse the child, who took a bottle with difficulty and vomited readily. The infant's tongue and abdomen became large, and her voice became hoarse; she was inactive and often constipated. She was taken to an infant welfare station, where she was given thyroid regularly from the eighth month. However, the mother became concerned when the child was not sitting up at 16 months of age and took her to a city hospital, where glandular treatment was prescribed and continued until the fourth year. Dentition began when the patient was 1½ years old and talking when she was 4 years old. When she came to the clinic at the age of 5½, her hair was normal but her skin was cold and dry. The thyroid gland was not palpable, and the fingers were short and stubby. A psychologic test at this time showed her to have an intelligence quotient of 61. The treatment was resumed; she showed marked improvement, and when the family moved to another state arrangements were made for medication there. On the family's return the following year, the pediatrician found the child had made decided physical progress.

The patient is now an attractive 8½ year old girl, 46½ inches (118 cm.) tall and weighing 47¼ pounds (21.5 Kg.). When she was 8 years old her intelligence quotient was 71. She has a pronounced stutter. Her appetite is good, but recently

she has been restless during her sleep, crying and grinding her teeth; she has reported crying when she dreamed her mother's head was cut off. Until a year ago she was enuretic. She bites her nails constantly. She likes dancing, singing, playing the piano, drawing, cutting and playing rough games. She began school when 5 years of age, remaining in the kindergarten one and a half years; at present she is repeating the first grade. She has learned little in school and shows no embarrassment over her slowness or the fact that her sister is doing better work. However, the latter is annoyed by the patient's "silliness" both at home and at school, where she continually grins, makes faces and hides behind people. The patient's schoolmates call her "dummy," and she says that the girls do not like her because they do not know her name. The mother states that the child gets along well enough with those outside the home but that she prefers to "parade" the streets by herself. There is evidence of a strong rivalry with the older sister, whom she has threatened to cut with a knife, and with the baby, whom she has pushed off the bed and dragged on the floor. She frequently accuses the parents of liking the baby better and has said that her sisters are mean to her; her one wish is that her dresses might be prettier than theirs. The mother, who does not realize the significance of the patient's behavior, believes that she has spoiled the child. She thinks that the patient has made slow improvement with the medical treatment and hopes that the child will be able to finish school. She no longer feels bad about the child's condition, as she is not now continually questioned about it.

Comment on Group II.—In general, the children in this age group have sleep disturbances which may indicate some emotional conflict and have difficulty relating themselves to their friends and siblings. These children, who seem to have less security with their parents, as evidenced by the parents' overprotection or rejection, are probably for that reason less able to get along with other children. With the exception of patient 11, low intelligence does not seem to be a factor in the difficulty in getting along well with friends, siblings and parents, since those children who are making a fair or relatively good adjustment do not differ materially in intelligence from the children making a poor adjustment.

GROUP III: PATIENTS TWELVE AND A HALF THROUGH SIXTEEN YEARS OF AGE

There are 8 patients in this age group. The following tabulation presents the sex, age, intelligence quotient and formal education of each:

Serial Number of Patient	Sex	Age	I. Q.	Education
16	F	12½	43	No attendance at school
17	F	13	48	No attendance at school
18	F	13	49	Few weeks
19	M	13	20	No attendance at school
20	M	13	70	6B grade
21	F	14	20	Few weeks
22	M	15	52	Special room
23	F	15	54	4B grade

All of these children had hypothyroid characteristics when first seen and with the exception of patient 17 have made remarkable physical improvement since receiving treatment. Patient 19, who has been receiving thyroid for the shortest period, is the only one now exhibiting

any signs of thyroid deficiency, and these are rapidly disappearing. Patient 16 is a deaf-mute; patient 17 has infantile speech, and patient 23 has strabismus. These children, who were all dull looking, apathetic and inactive, have become more alert and energetic; patient 23 has shown the least improvement in this respect.

Patients 16, 18, 19, 20, 21 and 23, who have made the best social adjustment, have good appetites and sleep well, though patient 19, the only one who is enuretic, has peculiar fears. Patients 16 and 18 bite their finger nails. All are able to dress themselves. All have similar play interests: The girls enjoy dolls, quiet games, motion pictures and the radio, while the boys prefer more active recreation. Patient 19, who lives on a farm, is especially interested in animals. Patient 18 is the only one who helps with household duties. On the whole, these children get along fairly well with their playmates, though the latter are limited in number. Patient 23, who would like friends, has none whatever and is teased by the neighborhood children and adults because she is so slow. Four of the 6 children have gone to school, but 2 were taken out after a short time because of lack of progress. Patient 23 dropped out of school at the fourth grade, and patient 20, who has an intelligence quotient of 70, is the only one who has made a relatively good adjustment in school. He is retarded but has been doing average work and was recently promoted to the 6B grade. The parents seem to have accepted the physical and mental problems of their children fairly well, the parents of patients 16, 18 and 19 appearing to have made a normal adjustment to their children. The latter are accepted by their siblings and seem to have few difficulties at home. The mother of patient 20 does not appreciate his school achievement because he does not come up to the high standards set by his sisters; she is overly protective and allows him little freedom. The mother of patient 21 accepts her child's retardation but regrets the girl's inability to help about the house, and the mother of patient 23 is overly protective and "babies" her daughter. All the parents feel that the children have made remarkable physical improvement since they have been receiving glandular therapy. The parents of patient 19 are proud of the fact that their son is now able to wear a ready-made suit; before treatment he was so misshapen that his clothing had to be made to order.

The patient dealt with in the following case report seems to be typical of the children in this age group who are making a relatively good social adjustment:

PATIENT 20.—The patient is a 13 year old boy, the youngest of 4 children. His father, born in Czechoslovakia, is one of 5 children; there is no history of a thyroid disorder in his family. The father went to school until he was 8, after which he did farm work until he came to this country at the age of 17. The mother was

one of 8 children of a poor family living in the same community as the father; there is no history of thyroid disorder in her family. She received eight years of education and came to this country when 14 years old to make her home with relatives. The father now has a goiter, and the mother says that she has one, although there has been no medical diagnosis. The mother supported the family with the assistance of the patient's sisters during the father's five years of unemployment and, though he recently secured work as a cabinet maker, she continues to work as a scrubwoman. Both grown sisters completed two years in high school, and one does clerical work while the other is employed as a maid. The older brother is doing passing work in the second year in high school. The family, which lives in a clean but sparsely furnished flat in a Bohemian neighborhood, has never been known to any social agency.

The mother was 29 and the father 27 at the time of the patient's conception. The normal delivery was made by a midwife. The patient was breast fed for one year; dentition began when he was 2 years old, and he started walking at the same time but was bowlegged. Toilet habits were not established until he was nearly 3 years old. When he was 4 years old his mother noticed that he was not developing properly, for he did not talk, looked sick and did not grow. She worried about his condition, but friends assured her that he was merely slow. It was a school nurse who finally referred him to the clinic when he was 6 years old. At that time he was a fat, undersized boy who weighed 37 pounds (16.8 Kg.) and was 27 inches (68.58 cm.) tall. His skin was dry and his features coarse. He had carious teeth, a large tongue and a distended abdomen. His movements were clumsy; his gait was waddling. A roentgenogram of the wrists showed evidence of healed rickets and of delayed ossification. His intelligence quotient was 50. Treatment was initiated, and his appearance changed rapidly. He grew taller, became more alert and began to talk. After one and a half years of medication he started to school. The mother discontinued medication after three years. The child's hair and skin again became dry; he was sluggish, indifferent and irritable. The mother then acknowledged that he was much healthier when taking thyroid and permitted treatment to be resumed. At the age of 10 years the patient underwent an operation for undescended testicles.

The patient is now a tall, slender, 13 year old boy, 60¾ inches (154 cm.) tall and weighing 99 pounds (45 Kg.). He has a serious expression, curly brown hair, large brown eyes and a ruddy complexion. He resembles his mother in that he has the same broad face and high cheek bones; his ears are unusually large and protruding. When he was 12 years old his intelligence quotient was 70. He eats well, has no sleep disturbances and is not enuretic. He plays with small toys, enjoys watching ball games and likes to fish and swim. He has several close boy friends and one girl companion. He entered kindergarten when he was 7 years old and was passed every year; recently he was promoted to the 6 B grade. During the past year he has received "average" in a large percentage of his subjects, with an occasional "excellent" and with "poor" in physical education. His mother believes that he is capable of doing better and compares him with his siblings, who received "superior" in all their subjects. The teacher described the patient as a "very good boy," but the mother says that all of her children behave well in school. The patient gets along well with his siblings, especially his older brother, of whom he is very fond, but he has the most fun with his father, who takes him fishing, although it is the latter who disciplines him. The mother thinks that the patient has been spoiled because of his physical condition. The parents have made no definite plans for his future but would like to have him attend high school if he is able.

Patients 17 and 22 have made the poorest adjustment of any of the children in this age group. Both sleep well, but patient 22 is afraid to retire without his brother and has incontinence of urine and of feces. He has a poor appetite and eats little but meat, whereas patient 17 eats well. Both children have play interests similar to those of the others in this age group. Patient 17 does not care for girls, but gets along satisfactorily with them; patient 22 teases the younger children with whom he plays and is ignored by the older boys. Patient 17 has never gone to school, and patient 22 is having difficulty in a special room. The mother of patient 17, a suspicious person, is overly protective of her daughter, seldom permitting her to do anything for herself and giving her little opportunity to learn the things a child of her mental age is capable of doing, while the father, who is not in the home, is not so solicitous and is critical of the mother's attitude; patient 22 seems to be accepted by his parents, but there is an unusual amount of rivalry among the siblings, which causes friction in the home. The mother of patient 17 thinks that the thyroid therapy has done her child more harm than good, and she has often discontinued the treatment, though the father thinks that the girl has shown improvement; the parents of patient 22 are satisfied with their son's physical progress.

The patient in the following case report is an example of children in this age group who have difficulty in making an adequate social adjustment.

PATIENT 22.—The patient is a 15 year old boy, the older of 2 children. His father was born in England and is one of 3 boys in a family in which there is no history of a thyroid disorder. He completed the eighth grade, came to this country when 21 years old and finally worked as a boilermaker. The mother was born in the Middle West and is one of 2 children. There is no history of a thyroid disorder in her family. She was a sickly child and had chorea at the age of 11 years. As a student she made little progress; her teachers always considered her slow. Until 1928 the patient's family lived in good circumstances, after which the father's work gradually slackened, and it has been necessary for the family to receive help from the public relief agency for the past four years. The mother is the stabilizing influence in the home; she is a calm, reasonable person who seems to have adjusted easily to their situation. The 13 year old brother is in the seventh grade in school. The family recently moved to a small, dark flat in a business district. Two social agencies have had contact with the family.

The mother was 29 and the father 36 at the time the patient was born. The mother was in a run-down condition; the labor was long, and she was torn during the delivery. At birth the patient had a large head and was "black." His mother nursed him until she became pregnant with the brother, although the patient drank from a cup when he was 10 months old. Dentition and talking began when he was 11 months old, but he was unable to walk until he was 19 months old. Though he was short and heavy, the mother thinks that he was normal until he was 2 years of age. When the family moved to the city a doctor made a diagnosis of rickets, and during the years that followed the patient was taken to many doctors, chiropractors and clinics. He was given thyroid from the

fifth through the seventh year, with little success. He was 10 years of age when he came to the clinic, but looked like a 6 year old child; he was 46½ inches (118 cm.) tall and weighed 57½ pounds (26 Kg.). He was fat and had a pasty complexion, stiff coarse hair and a dry skin. The abdomen protruded and the voice was deep. He had both diurnal and nocturnal enuresis. A mental examination at this time showed that he had an intelligence quotient of 42. The treatment was initiated, and the boy gained in height and weight. He was nervous at times but seemed better able to care for himself. The enuresis was less frequent, and his mother thought he was "getting smarter."

The patient is now a rather attractive 15 year old youth with light brown hair, blue eyes and a fair complexion. He is 62½ inches (158.75 cm.) tall and weighs 97¼ pounds (44.13 Kg.). When he was 14 years old his intelligence quotient was 52. He is a shy boy, and his mother describes him as "happy go lucky." He has a poor appetite, eats little except meat and dislikes sharp or bitter tastes. He is afraid to retire before the brother, and the latter continually complains of the patient's bed wetting, which the mother has made many unsuccessful attempts to correct. The patient has shown a marked interest in mechanical devices and spends much time watching his father do repair work. Since receiving the treatment he has been more active and interested in playing, but he refuses to enter into rough games for fear of being hurt. He does not like to read or listen to the radio, and though he goes to motion pictures occasionally, he does not understand them. He attends Sunday school regularly and takes part in the various programs, but he has difficulty in sitting still, which disgusts the brother. The patient usually plays with younger children, with whom he gets along fairly well, though he teases them. The older boys either ignore him or take advantage of him, but recently he has been asserting himself. He started to school at the age of 10 years and at present is doing poor work in a special room. He dislikes his teacher, is frequently tardy and urinates in class. He is mistreated by the other children. He has been such a problem that the teacher has threatened to exclude him from school.

When he and his brother were younger the patient's retardation did not interfere with their relations, for the brother fought the patient's battles and the children were good friends. Now, in spite of the mother's admonitions, the brother teases the patient, and quarrels ensue. The brother is ashamed of the patient and resents the parents' favoring him. Now that the patient is becoming stronger physically the mother has difficulty disciplining him and has found that withholding privileges is most effective. The patient gets along very well with his father, though he minds him no better than he does his mother. When matters do not go right the patient becomes angry and wishes he were dead. He has used his mental retardation as an excuse for not doing things, though the mother has attempted to motivate him by telling him that he is grown up. The parents think that his physical progress has been "wonderful" but that he has shown little improvement mentally. The mother regrets they were unable to secure medication sooner. The parents realize that the boy can learn to care for himself only by working with his hands and hope that with continued treatment he will be able to attend an industrial school.

Comment on Group III.—The children in this age group, despite the fact that on the whole their intelligence is lower than that of the preceding age groups, seem to have less difficulty in their social adjustments, although their relationships to other children appear restricted. Their

relatively good adjustment may be related to their greater security with their parents or to the fact that, with the exception of patient 20, whose general school adjustment is good, they are no longer competing with their superiors.

GROUP IV: PATIENTS SEVENTEEN THROUGH TWENTY-ONE YEARS OF AGE

Serial Number of Patient	Sex	Age	I. Q.	Education
24	M	17	69	7th grade
25	F	17½	62	8th grade graduate
26	M	18	39	1st grade
27	M	21	83	2 yr. of high school

These children when first studied were dull, apathetic and uninterested in their surroundings. Under treatment they have improved in physical condition, but patients 24, 25 and 26, who have the lowest intelligence quotients, still look dull. Patient 27, who has the highest intelligence quotient, cannot be recognized as having a physical handicap. Patient 26, who has made the slowest progress, still stutters severely. All of the children have become more alert and active, patient 26 remaining the most infantile.

Patients 24 and 27, who have made the best social adjustment, eat and sleep well and have a variety of recreational interests, such as reading, listening to the radio, playing games and attending motion pictures. Patient 24 has few friends, and although patient 27 is popular with those of his own sex he has limited contacts with the opposite sex. Patient 24 finished the seventh grade, and patient 27 attended high school for two years. Patient 24 does gardening and helps his mother with the housework, and patient 27 is in a C.C.C. camp, where he earns $30.00 a month, which pays his parents' rent. Both seem to be accepted by their families. The parents of patient 24 have pushed their son academically, perhaps because the mother is a school teacher, but they realize his intellectual limitations; the mother of patient 27 tries to be understanding and undoubtedly is motivated by her feelings of guilt because she tried to abort him. The parents of both children are satisfied with the treatment.

The patient of the following case report is fairly typical of the children of this age group who are making a satisfactory social adjustment.

PATIENT 27.—The patient is a 21 year old Jewish boy, the sixth of 7 children. His father, born in Russia of a family in which there is no history of thyroid disorder, received a limited education and then followed the blacksmith trade. By a first marriage he had 4 children, all of whom are in good health. The mother, twelve years his junior, was also born in Russia, and there is no history of a thyroid disorder in her family. She received little formal education, starting to work at an early age. The patient's parents were married after the death of the father's first wife, and his 4 children were reared with their 7. They were very

poor in Russia; the children received little education and were often without proper clothing. The family came to this country twenty-six years ago in the hopes of bettering their living conditions, but they have always been in straitened circumstances; for the past twenty-one years it has been necessary for the father to secure loans from a charitable organization or to receive financial assistance from one of the eight social agencies with which they are registered. At present the mother supplements the father's meager earnings as a blacksmith by working in a laundry, and the $30.00 which the patient earns at the C.C.C. camp pays the rent. All of the children except the patient and a younger brother are married and have families. The patient is the only one who was not graduated from high school. The family resides in a Jewish neighborhood in the rear of a rundown apartment building, but the flat is clean and attractively furnished.

The mother believes that she is responsible for the patient's hypothyroid condition, for she tried to abort him. She was well during the pregnancy, but the labor was long. The patient weighed 9½ pounds (4,309 Gm.) and had difficulty in breathing at first. His mother nursed him for eleven months. His development was slow, for he sat up at the age of 8 months and walked and talked at 2 years of age and dentition had not started when he was 3 years old. He used to sit in a chair all day showing no interest in the other children's play. He was 9 years old when he came to the clinic and at that time was 50½ inches (128 cm.) tall and weighed 39½ pounds (18 Kg.). He was fat and his skin was dry and scaly and his hair thin. He had a large abdomen, an umbilical hernia and lumbar lordosis. There was evidence of mild rickets. The treatment was initiated, and the patient soon lost the hypothyroid characteristics. Medication continued for twelve years, the patient coming to the clinic regularly. At times he seemed to be nervous, but the mother made no complaints as long as he was receiving glandular therapy. At 17 years of age he was given his first mental test and was found to have an intelligence quotient of 80. Two tests were given in the next two years with practically the same results. To all appearances he was a normal boy physically. He enrolled in a C.C.C. camp when he was 19 years old, and it was after this that secondary sex characteristics developed.

The patient, who is now 21 years old, is 61¾ inches (157 cm.) tall and weighs 124 pounds (56 Kg.). He is slender and well built, with straight dark hair, brown eyes and regular features. He dresses neatly and in good taste. He seldom complains about anything, and if worried he writes to his married sister rather than disturb his mother. He has never been an affectionate boy. He eats and sleeps well and can care for all of his bodily needs. He has always liked to play ball, but has been good about helping his mother at home. He had many friends when he was living in the city, the boys frequently coming to him for advice. Although he said he had no interest in girls, he suspected that they laughed at him because he was short, and he disliked going to dances for this reason. Before his secondary sex characteristics developed he refused to swim at school, and the pediatrician, who felt that the boy was beginning to have an inferiority complex, attempted to help him overcome this feeling.

The patient started to school when he was 6 years old, but did not attend regularly at first because he was afraid. His work was always poor, and he was forced to repeat several grades, so that he was 17 years old when he entered high school for a general course. During his first year in the secondary school he was given a number of achievement tests and was found to be deficient in subjects requiring abstract reasoning. He had a placement of 4.8 in both arithmetic reasoning and computation, 5.4 in reading comprehension and 6.7 in word meaning, 7.5 in language, 6.6 in literature and 8.1 in history and civics. As he disliked being so

much older than the other students and felt that he should be working when the family had so little money, he left school in his second year of high school. He made an earnest effort to secure work and finally was given a temporary job in a fruit store. Shortly afterward he enlisted in a C.C.C. camp, where he has been for the past two years. The clinic nurse has corresponded with him during the last year; he writes short, uninteresting letters in a childish scrawl, misspelling many words. So far as can be ascertained he is happy and enjoying his experience.

Patients 25 and 26 seem to have made the poorest adjustment in this age group. They also have the lowest intelligence quotients, 62 and 39 respectively. Both have good appetites and sleep well, but patient 26 is still enuretic. Patient 25 has few interests besides reading, embroidering and going to church, and the interests of patient 26 are infantile but in keeping with his mental age. Both have few friends, and patient 25, who has been teased by girls of her own age, prefers to play with younger children, although her opportunities are limited; patient 26 has not been accepted by other children because he is so retarded mentally. Patient 25 has been slow in school, but with pressure from her parents and cooperation from the school, she was permitted to be graduated from the eighth grade, although it was known that she unable to do work at this level; patient 26 attended school for only a few months. Both children can perform household duties, but patient 25 is the more responsible. At one time the latter's parents refused to take her anywhere because they felt disgraced, and her siblings refused to have anything to do with her, saying she was "dumb;" her mother is still unable to accept her mental retardation and is critical of her behavior. Patient 26 has never been accepted by his siblings, who resent having to play with him. The parents of both children have been satisfied with the treatment, although the mother of patient 25 does not give her the thyroid regularly, believing it makes her nervous.

The patient in the following case report is an example of children in this age group who have made a poor social adjustment.

PATIENT 26.—The patient (no. 26) is an 18 year old boy, the third of 7 children. His father was born in Austria and is 1 of 7 children of a family in which there is no history of a thyroid disorder. He received a fourth grade education and came to this country at the age of 19, settling in a small mining town in the central part of the state, where he has worked for a railroad for many years. The mother, who is fifteen years the father's junior, is one of 10 children of German parents. She worked in a laundry after going through the fifth grade in school. She married the father when she was 18. After the birth of the youngest sibling a goiter developed in the mother, although there has been no history of a thyroid disorder in her family. It is reported that she is a conscientious mother and housekeeper. All of the children are in good health with the exception of a brother who has multiple osteochondromas. The family has been known to two social agencies.

The mother was 20 and the father 35 at the time of the patient's birth. The former was nursing the sister while pregnant, and she thinks that this might have

caused the patient's condition. The patient weighed 8 pounds (3,629 Gm.) at birth. He was breast fed for one and a half years and had difficulty in eating solid foods because of his large tongue. Dentition began when he was 1½ years old and walking when he was 3 years old; he was able to say only a few simple words at the age of 5 years. The delay in talking caused the mother to seek medical advice. His condition was diagnosed as hypothyroidism by the family physician when the patient was 2 years old, and thyroid was administered for the next eight years. The doctor then lost contact with him. When the child was 12 years old the parents applied to a child guidance clinic for advice regarding his supervision. He was found to have an intelligence quotient of 48, and commitment to a state institution for the feebleminded was recommended, as the mother seemed unable to give him the necessary attention. It was at this time that he was referred to the clinic for treatment. Although he was 12 years old, he looked like a child of 7 years, being 49¼ inches (143 cm.) tall and weighing less than 45 pounds (20.5 Kg.). He was short, and his skin was dry and his forehead wrinkled. He had a large, protruding tongue. His face was old appearing. He had a scrotal hernia. His carpal development was that of a 5 year old child. His speech was coarse; he stuttered severely and talked little. He refused to play, but otherwise was cheerful and obedient. He was admitted to the hospital, where he remained for three months, during which thyroid was administered and the scrotal hernia was repaired. He became "completely transformed," for he grew taller, lost weight and seemed more alert. The following fall he started to school and was mischievous; his mother commented, "He didn't have sense enough to be, last year." Stuttering and enuresis persisted, but the child became more active and played with other children. Five years after the beginning of this second period of treatment his secondary sex characteristics developed.

The patient is now a rather dull looking 18 year old boy with brown eyes and light brown hair, who appears younger than his age. He is 60½ inches (154 cm.) in height and weighs 94 pounds (42.5 Kg.). His mouth waters readily; he stutters, and he has severe facial spasms when he attempts to talk. His motor coordination is poor. At the age of 15 years and 10 months he was found to have an intelligence quotient of 39. He is never cross and minds well. He is able to care for his bodily needs and is always neatly dressed. He sleeps well, sometimes having "nice" dreams about Easter eggs and candy, and he is less enuretic than formerly. He has a good appetite and "eats everything." He likes to help his mother do the housework and can be trusted to go to the store. When not working, he colors pictures or plays with his youngest brother. He enjoys hunting snakes in the woods and likes to make such things as bird houses. He is too slow to play baseball, and he uses roller skates with difficulty. His mother does not let him play much with boys, and most of his companions are younger children, all of whom like him. He is afraid of his brothers and feels resentful because they do not take him places, although the mother insists that they do so occasionally, as she thinks he should be with older boys. The patient has said that he is afraid of children because they beat him up, and he has fantasies about giving various boys bloody noses. He started to kindergarten when he was 11 years old, and after receiving treatment he entered the first grade at the age of 12½ years, attending for a few months. He was never able to learn, and finally the mother stopped sending him to school when he cried because the boys teased him. Recently he has mentioned wanting to go again.

The siblings have never wanted to play with the patient, and the latter continually complains that the 10½ year old brother "picks on" him, especially when he wants to be taken somewhere. The patient thinks his 14 year old sister is

"pretty good" since she is never cross with him, but he likes his baby brother best. He thinks that the father also "picks on" him but the mother is "nice." He says that he is seldom happy, for his siblings will not take him anywhere, and he attributes this to the fact that they think he is dirty. The mother used to be upset because of the patient's condition, but she worries less now that he has shown so much physical improvement, is more observing and acts more like other children. The older sister believes that the patient's siblings are more willing to play with him now. The parents have made no plans for his future.

Comment on Group IV.—Two of the 4 children in this age group have made a relatively satisfactory adjustment. Low intelligence coupled with rejection by parents and siblings seem to be important contributing factors to the maladjustment of the other 2. There has been more academic success among the children in this group than among those in the other groups, although it has taken these patients longer to complete specific units of work than it would take the normal child. One of the children has made a relatively successful work adjustment.

SUMMARY

The purpose of this study was to examine the background and the social adjustment of hypothyroid children who have received glandular therapy for several years. The following observations were made:

The children studied come from smaller families than those of their parents. They have a wide range in intelligence, extending from very defective to average, the majority tending to be in the high grade mental defective group. These children have been able to establish habits in accordance with those of normal children of the same mental age, and except when they have been restricted by overprotective parents, their play interests are similar to those of normal children.

The patients between the ages of 1 and 7 years and those of 17 through 21 years seem to have made the best social adjustment and are the best accepted by their parents. The acceptance of the youngest group may be due to the fact that they are still considered babies and are sheltered in the home; their relatively good social adjustment may be fostered by their security with their parents. The acceptance of the oldest group by their parents may be due to the fact that they are able to take care of themselves and cause little trouble; their relatively good social adjustment may be facilitated by their security with their parents and by their successful experiences in school and work.

The children having the most difficulty in adjusting to other children are those between the ages of 7 and 12 years. This may be due to the fact that they are no longer sheltered as are the younger children and they are making their first attempts to relate themselves to normal children and to the school situation. There is more maternal rejection and sibling rivalry in this group than in any of the other groups. Possibly

the parents are beginning to expect the patients of this age to do the things normal children of the same age do, and they may become impatient when the children fail and are a care, for they have no assurance that these atypical children will develop into independent adults. The children between the ages of 12 and 17 have made a better adjustment than those in the younger group, but have not been accepted at their level by their parents as well as have the older children. There is some maternal overprotection and sibling rivalry in this group.

Glandular therapy has been effective in helping these children to make a better social adjustment. It has accelerated their physical growth, stimulated them to activity and caused them to become more normal in appearance. They have been able to establish toilet habits and to develop play interests that probably would have been impossible without medical treatment. They seem to be better accepted by other children and by their parents, partly because their appearance is more normal and partly because their behavior has become more like that of children of the same chronologic age. The adjustment of the older group suggests that even with low intelligence, children with thyroid deficiency are able, with treatment, provided there are healthful attitudes toward them within the home, to be partially self-supporting, to be helpful in the home and to work out satisfactory relationships in the family. However, glandular therapy does not overcome low intelligence or unwholesome parental attitudes, and an improvement in parental attitude does not always accompany physical improvement in the child.

Hattie Larson, R.N., rendered valuable assistance in this study.

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TRANSITORY SYNOVITIS OF THE HIP JOINT IN CHILDREN

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Anomalies of the hip joint in children are common enough to warrant a better understanding of them by the general practitioner and pediatrician. This is especially true of one of the benign forms, to which the term transitory synovitis of the hip joint has been applied. Some of the previous presentations on this condition will be reviewed in this paper, and the cases of 37 patients observed by me during the past two and one-half years (January 1937 through June 1939) will be analyzed and discussed.

From the contributions of Belmonte,[1] Butler,[2] Todd,[3] Fairbank [4] and Finder,[5] who described the same syndrome under a variety of closely related titles, the disease has come to be regarded as a nonspecific, relatively transient inflammation of the synovial membrane of the hip joint of a child. The onset may be acute or insidious. Clinically, there are symptoms of pain, limp or unwillingness to walk and signs chiefly of restriction of hip motion. Laboratory evidence of infection (fever, leukocytosis, rapid erythrocyte sedimentation) is usually lacking but may be present. The intradermal tuberculin reaction is, with an occasional exception, negative, and the roentgenogram of the hip joint invariably so. The treatment is conservative and the prognosis for functional recovery excellent.

When in 1931 Belmonte [1] described the clinical picture as that of a transitory benign coxitis, he had several objects in view: (1) to differentiate the disorder from tuberculous coxitis; (2) to point out its relative frequency, although no figures of incidence were mentioned, and (3) to focus attention on the possibility of diagnosing it as a clinical entity

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2. Butler, R. W.: Transitory Arthritis of the Hip Joint in Childhood, Brit. M. J. **1**:951, 1933.

3. Todd, A. H.: The Differential Diagnosis of Non-Tuberculous Coxitis in Children and Adolescents, Proc. Roy. Soc. Med. (Sect. Orthoped.) **18**:31, 1925.

4. Fairbank, H. A. T.: Discussion on Non-Tuberculous Coxitis in the Young, Brit. M. J. **2**:828, 1926.

5. Finder, J. G.: Transitory Synovitis of the Hip Joint in Childhood, J. A. M. A. **107**:3 (July 4) 1936.

at the very outset. He also quoted the names "coxitis serosa seu simplex," "fugitive or ephemeral coxitis," "coxitis fugax" and "coxitis of uncertain cause" employed by other writers in relation to the syndrome. Butler,[2] in 1933, in discussing the group of "observation hips," described 22 cases of transitory arthritis of the hip joint, in 19 of which there was complete return of function within an average period of three years. He indicated that at one time these instances of recovery might have been regarded as "cures" of tuberculosis. In considering the question of the cause of this transitory arthritis he expressed the opinion that an infective process was the most likely causative factor, probably a transient epiphysitis never sufficiently localized and well defined to show changes in the bone in a roentgenogram. Todd[3] included the syndrome in a discussion of the differential diagnosis of nontuberculous coxitis in children and adolescents. Under the heading of "traumatic coxitis" he spoke of "simple transient coxitis," but recommended "contusion of the hip" or "synovitis" as a preferable term. He also mentioned the infective forms of coxitis consequent on acute febrile conditions, some microbic and some toxic, as well as the coxitides secondary to acute or subacute epiphysitis. Fairbank[4] also classified nontuberculous coxitis in the young, and in his list were traumatic synovitis and arthritis in addition to infective arthritis. In 1936 Finder[5] reported 22 cases, and logically, on the basis of the pathologic changes, designated the disease as transitory synovitis rather than as transitory arthritis. He was careful to point out that symptoms of transitory synovitis of the hip joint in children may be simulated closely by the early stage of other forms of disease of the hip joint in which synovitis is the initial lesion. On studying the pathologic process in 1 of his patients with disease of the hip joint of longer standing, who was suspected of being tuberculous and on whom surgical intervention was employed, Finder observed only a chronic synovitis of the hip joint with collateral osteoporosis. He formulated the concept that when local resistance and immunity of the synovial tissue are adequate the infection subsides, and that transitory synovitis constitutes the entire disease; that on the other hand, unfavorable defense permits the infection to develop from the synovitis into the arthritis of any one of several chronic and relatively malignant disorders of the hip. With this simple and logical point of view I am in accord. However, since the course of events cannot be determined except after the passage of an indefinite, even though short, time, I prefer to make Butler's[2] less hazardous diagnosis of "observation hip," namely, probably transitory synovitis of the hip joint. In other words, the diagnosis should be one of exclusion, to be determined in two stages: (1) the stage of ruling out in a few days by laboratory procedure all other possible forms of hip disease, and (2) that of observing the patient for several years to see that the disease was not only benign but transitory.

Before turning to the cases to be presented, it is well, in order to avoid controversy, to consider the cases of acute transient epiphysitis of the hip joint described by Miller [6] in 1931. This group of cases very closely resembles that being described as cases of transitory synovitis. However, the 77 cases reported are by definition dissimilar, the distinction between epiphysitis and synovitis being demonstrated by roentgenograms. In instances of the former condition, even though first roentgenograms may show no pathologic signs, at some time during the period of observation changes in bone texture or joint outline, localized abscesses or areas of absorption along the epiphysial line in the femoral neck or head become manifest. The roentgenograms in instances of transitory synovitis repeatedly show normal conditions. One can reconcile, then, the possibility of the existence of these two conditions side by side without assuming that in the cases of synovitis changes are not seen in roentgenograms either because the roentgenograms are erroneously interpreted or because the lesions had healed and become undetectable at the time the roentgenograms were taken.

Ferguson and Howorth,[7] in their analysis of coxa plana (Legg-Calvé-Perthes disease) and related conditions of the hip, approached the problem of pathologic changes from another point of view. They expressed the opinion that these disorders of the joint result from a circulatory disturbance in or adjacent to the femoral head. Their arrangement of five types of ischemia follows:

Type 1. Inflammation in the hip joint.

Type 2. Inflammation or, rarely, tumor adjacent to the joint, usually in the femoral neck.

Type 3. Fracture or epiphysial separation at the femoral neck.

Type 4. Trauma to the femoral head.

Type 5. Tension of the soft tissues about the femoral neck.

They found that in the cases in which coxa plana appeared earliest

"the presence of extensive changes in the synovial membrane, capsule, and periosteum, before the contour of the head had become abnormal indicated that these changes are not secondary to trauma caused by a deformed head, but are primary. They apparently are the result of previous synovitis or arthritis, probably infectious, and they appear to be the cause of the subsequent changes within the epiphysis through the medium of the circulatory disturbance which they produce, the Type 1 ischemia. . . ."

6. Miller, O. L.: Acute Transient Epiphysitis of the Hip Joint, J. A. M. A. **96**:575 (Feb. 21) 1931.

7. Ferguson, A. B., and Howorth, M. B.: Coxa Plana and Related Conditions at the Hip, J. Bone & Joint Surg. **32**:781, 1934.

TABLE 1.—*Summary of Thirty-Seven Cases of*

Case, Name; Sex, Color	Age, Yr.	Date of First Entry	History Preceding Onset of Chief Complaint: Illness or Operation	Fever (Highest Temperature, F.)	Injury	Interval Before Onset of Chief Complaint, Days	Extremity Involved	Physical Examination: Pain	Limp	Duration of Chief Complaint Before Entry; Date of Onset	Resistance to: Abduction	Resistance to: Extension	Resistance to: Internal Rotation	Atrophy
1. N. G.	(a) 5	1/17/34	0	0	0	0	L	Groin	+	?	+	+	+	0
M, W	(b) 8	1/19/37	0	0	0	0	L	Thigh	+	?	+	+	+	0
	(c) 8½	8/ 3/37	0	0	0	0	R	Right thigh and knee	+	2 days 8/1/37	+	+	+	0
2. L. G.	(a) 8	2/../35	0	0	0	0	R	+	+	?	?	?	?	?
M, W	(b) 10	8/19/37	0	0	Blow from hammer	1	R	+	+ (Intermittent)	14 days 8/5/37	+	+	+	0
	(c) 10	10/16/37	0	0	0	0	L	0	+	3 days 10/13/37	+	+	+	0
3. W. L.	(a) 11	1/19/37	0	0	0	0	L	+ (Intermittent)	+	?	+	+	+	0
M, W	(b) 11	4/15/37	Common cold	100	0	7	R	+	+	7 days 4/8/37	+	+	+	0
4. M. S. M, W	5	3/ 4/37	Bilateral otitis media	102	0	75	L	+	+	2 days 3/2/37	+	+	+	0
5. I. Q. F, W.	11	3/24/37	0	0	0	0	R	Right thigh and knee	+	60 days 1/23/37	+	+	+	0
6. J. M. M, W	7	3/25/37	0	0	Fall	1	R	Right groin (intermittent)	+	30 days 2/23/37	+	+	+	0
7. I. R. M, W	6	4/ 9/37	Common cold	100	0	10	R	Right lower abdomen and groin	+	3 days 4/6/37	+	0	+	0
8. S. L. M, W	8	5/ 3/37	0	0	0	0	R	0	+	9 days 4/24/37	+	+	+	0
9. P. R. F, W	4	6/22/37	Pneumonia with pleural effusion	104	0	90	R	+	+	85 days 3/29/37	+	+	+	0
10. M. V. F, W	10	7/10/37	0	0	0	0	L	Left groin	0	2 days 7/8/37	+	+	+	0
11. J. F. F, W	7	8/ 2/37	Sore throat	102	0	21	R	+	+	14 days 7/19/37	+	0	+	0
12. M. D. M, W	7	8/ 6/37	0	0	Fall from bed	7	R	Right knee	+	7 days 7/31/37	+	+	+	0
13. J. F. F, W	10	8/24/37	0	0	0	0	L	Left thigh	+	30 days 7/25/37	+	+	+	0

Laboratory Data								Course		
					Roentgenograms					
Old Tuberculin Test	Sedimentation Rate of Erythrocytes	Highest Temperature (F.) After First Entry	Kline Test	Blood Count	Date	Interpretation	Follow-Up Roentgenograms and Interpretations	Treatment	Duration of Illness from Onset to Recovery, Days	Present Status	Comments
Neg.	.....	99	.....	.....	1/18/34	Neg.	.....	Rest in bed	?	Well	Not seen by me in first attack, but symptoms and signs suggest synovitis; recurrence with same symptoms and signs; personal history of decided allergy; heart normal; patient under observation
.....	.....	99.4	.....	.....	.....	.....	.....	Rest in bed, salicylates	?	Well	
Neg.	Normal	100.2	.....	.....	8/ 4/37	Neg.	.....	Rest in bed	16	Well	
.....	.....	?	.....	.....	.....	.....	.....	Rest in bed	?	Well	Was said to have had "dislocation" of right hip in 1935; recovered after rest in bed; may have been a synovitis of hip; has papular urticaria and asthma and is being observed and treated in allergy clinic
Neg.	Normal	99.2	.....	.....	8/20/37	Neg.	.....	Rest, baking and massage	30	Well	
Neg.	.....	99	.....	.....	10/20/37	Neg.	.....	Rest	21	Well	
Neg.	.....	100	.....	.....	4/10/37	Neg.	.....	Rest in bed	?	Well	Question of rheumatic fever entertained in April 1936, but heart and sedimentation test normal; intermittent pains in left thigh thereafter; history of frequent injuries; seen for trauma to back in August 1938 and to finger in February 1939
.....	Normal	99	.....	.....	.....	.....	.....	Rest in bed	21	Well	
Pos. to 5 mg.	.....	99.8	Neg.	.....	3/ 3/37	Neg.	.....	Rest in bed	90	Well	Was in hospital for otitis; had intermittent pain in hip for 2 mo. after discharge to home; limp noticed on first attempt at walking; also has allergic rhinitis and infectious asthma; mastoidectomy on March 26, 1937
Neg.	.....	100	Neg.	W.B.C., 11,000; poly. 76%	3/25/37	Neg.	.....	Bed, Buck's extension and cast for 1 mo.	120	Well	Treated in hospital
Neg.	.....	98.8	.....	.....	3/25/37	Neg.	.....	Rest in bed	60	Well	
Neg.	.....	99.8	.....	.....	.....	.....	.....	None	10	Well	Uncooperative; condition improved even without rest in bed
.....	.....	98.6	.....	.....	5/ 3/37	Neg.	.....	Rest in bed	?	?	Did not return for follow-up
Neg.	.....	99.4	.....	.....	6/23/37	Neg.	.....	Rest in bed, supinating heel braces for flat feet	100	Well	Had pneumonia with pleural effusion April 1, 1937; no operation or other possibilities suggested; muscular debility following long stay in bed, transitory epiphysitis; roentgenograms negative; child obese, but no evidence of hyperthyroidism
.....	.....	100	.....	.....	.....	.....	.....	Rest in bed	16	Well	Had angioneurotic edema and urticaria in 1935; frequent attacks of abdominal pain; heart normal; family history of rheumatism and allergy
.....	.....	100.4	.....	.....	8/ 2/37	Neg.	.....	Rest in bed, salicylates	?	?	Did not return for follow-up
Neg.	Normal	99.4	.....	.....	8/ 7/37	Neg.	.....	Rest in bed	14	Well	
.....	.....	98.6	.....	.....	8/27/37	Neg.	.....	Rest in bed, baking and massage	54	Well	

TABLE 1.—*Summary of Thirty-Seven Cases of Transitory*

Case, Name; Sex, Color	Age, Yr.	Date of First Entry	History Preceding Onset of Chief Complaint: Illness or Operation	Fever (Highest Temperature, F.)	Injury	Interval Before Onset of Chief Complaint, Days	Extremity Involved	Physical Examination: Pain	Limp	Duration of Chief Complaint Before Entry; Date of Onset	Resistance to: Abduction	Resistance to: Extension	Resistance to: Internal Rotation	Atrophy
14. G. K. M, W	2	8/25/37	0	0	Fall from tricycle	7	R	+	+	5 days 8/21/37	+	+	+	0
15. M. G. M, W	10	10/25/37	0	0	0	0	R	Right groin	+	1 day 10/24/37	+	0	+	0
16. M. Y. M, W	5	11/ 1/37	Common cold	99.2	0	14	R	Right thigh on awakening	+	1 day 10/31/37	+	+	+	0
17. D. N. M, W	2	12/16/37	0	0	0	0	R	+	+	120 days 8/18/37	Slight	Slight	Slight	0
18. D. B. F, W	9	2/17/38	Grippal infection	103	0	14	R	+	+	7 days 2/10/38	+	+	+	0
19. M. C. F, W	6	5/23/38	Measles Tonsillectomy	102 ...	.. .. Fall	34 18 1	R	Right thigh	+	1 day 5/22/38	+	+	+	0
20. D. C. F, W	5	7/ 9/38	0	0	Bump	14	L	+	+	3 days 7/6/38	+	+	+	0
21. L. F. F, W	1⅓	8/ 1/38	0	0	Fall down stairs	90	R	0	+	90 days 5/3/38	Slight	0	Slight	0
22. T. B. M, W	5	8/15/38	0	0	0	0	L	On awakening	+	1 day 8/14/38	+	+	+	0
23. S. Z. F, W	5	8/17/38	Measles Sore throat	103 102	0 0	60 30	L	Left groin after bath	+	1 day 8/16/38	+	0	+	0
24. E. B. M, W (a)	8	9/24/38	0	0	0	0	R	+	+	7 days 9/17/38	+	+	+	0
(b)	8½	2/18/39	Grippal infection	102	0	21	L	+	+	7 days 2/11/39	+	+	+	0
(c)	9	5/13/39	0	0	0	0	L	0	+	3 days 5/10/39	Slight	0	0	0
25. L. F. M, W	4	10/13/38	0	0	0	0	L	+	+	2 days 10/11/38	+	+	+	0
26. C. S. M, C	7	11/ 1/38	0	0	Fall	4	R	Right lower abdomen and groin	+	4 days 10/28/38	+	+	+	0
27. D. M. F, C	7	1/ 7/39	0	0	0	0	R	Right groin, lumbar spine	+	2 days 1/5/39	+	+	+	0
28. S. R. M, W (a)	7	3/20/39	0	0	Fall on pavement	2	L	+	+	2 days 3/18/39	+	+	+	+*
(b)	7	4/ 3/39	0	0	0	0	L	0	+	2 days 4/1/39	+	+	+	0
29. J. D M, W	9	3/23/39	Common cold	100	0	4	L	+ Left pubic area	0	10 days 3/13/39	+	+	+	0

Laboratory Data								Course			
					Roentgenograms						
Old Tuberculin Test	Sedimentation Rate of Erythrocytes	Highest Temperature (F.) After First Entry	Kline Test	Blood Count	Date	Interpretation	Follow-Up Roentgenograms and Interpretations	Treatment	Duration of Illness from Onset to Recovery, Days	Present Status	Comments
Neg.	.....	99	.....	.....	9/ 1/37	Neg.	.....	Rest in bed	?	?	Did not return for follow-up
Pos. to 5 mg.	Normal	99.2	.....	W.B.C., 8,500; polys. 47%	10/27/37	Neg.	.....	Rest in bed	15	Well	Observed as having possible rheumatic fever; history of abdominal and joint pains; torticollis in 1935; functional heart murmur many years
Neg.	.....	98.6	.....	.....	11/26/37	Neg.	.....	Rest in bed	8	Well	History of abdominal pain and intolerance to egg; grippal infection in March 1938; no evidence of rheumatic fever; familial history of allergy
.....	.....	99.4	.....	.....	.....	.....	.....	Rest in bed	?	?	Did not return for study; probably had subsiding synovitis when seen; diagnosis not definite
Neg.	Slightly increased, 35 mm. in 1 hour	98.6	.....	.....	2/17/38	Neg.	Neg. 4/1/38	Rest in bed	49	Well	Being observed as having possible rheumatic fever; heart normal
Neg.	Slightly increased, 25 mm. in 1 hour	99	Neg.	.....	5/23/38	Neg.	.....	Rest in bed	8	Well	Had measles, tonsillectomy and injury all before onset of hip disease; also had history of pyuria in 1935 and frequent abdominal pain
.....	.....	99.2	.....	.....	.....	.....	.....	Rest in bed	?	Well	Did not return for one year; no further complaint
.....	.....	100.2	.....	.....	.....	.....	.....	Rest in bed	97	Well	Hematoma of right thigh after fall; when seen in clinic signs at hip minimal; may have had traumatic synovitis
.....	.....	103	.....	.....	8/19/38	Neg.	.....	Rest in bed	8	Well	No evidence of rheumatic fever; heart normal
.....	.....	100.2	.....	.....	8/19/38	Neg.	.....	Rest in bed	8	Well	Obese child with papular urticaria and familial history of allergy
Pos. to 5 mg.	Normal	99.2	.....	.....	9/30/38	Neg.	.....	Rest in bed	21	Well	
.....	.....	99	.....	.....	.....	.....	Neg. 2/18/39	Rest in bed	14	Well	
.....	.....	99.4	.....	.....	.....	.....	.....	Rest in bed	10	Well	
Neg.	.....	99	Neg.	.....	10/14/38	Neg.	.....	Rest in bed	16	Well	Has hereditary ectodermal dysplasia
Neg.	.....	99	.....	.....	11/ 8/38	Neg.	.....	Rest in bed	?	?	Repeated attacks of abdominal pain; diarrhea in September 1938; did not return for follow-up; one of two colored patients in series
Neg.	Rapid, 88 mm. in 1 hour	99.2	Neg.	.....	1/11/39	Neg.	.....	Rest in bed	32	Well	Erythrocyte sedimentation very rapid; heart normal; spleen not palpable; the other of two colored children in series
Neg.	.....	98.2	.....	.....	3/20/39	Neg.	.....	Rest in bed	23	Well	Recurrence in same extremity of short duration; has vasomotor rhinitis
.....	.....	98.2	.....	.....	.....	.....	Neg. 4/14/39	Rest in bed	4	Well	
Neg	Normal	100	.....	.....	3/25/39	Neg.	Neg. 5/11/39	Rest in bed, physical therapy	70	Well	Painful finger 5 days before hip pain; no evidence of ischiopubic osteochondritis; heart normal

TABLE 1.—*Summary of Thirty-Seven Cases of Transitory*

Case, Name; Sex, Color	Age, Yr.	Date of First Entry	History Preceding Onset of Chief Complaint: Illness or Operation	Fever (Highest Temperature, F.)	Injury	Interval Before Onset of Chief Complaint, Days	Extremity Involved	Physical Examination: Pain	Limp	Duration of Chief Complaint Before Entry; Date of Onset	Resistance to: Abduction	Extension	Internal Rotation	Atrophy
30. M. H. F, W	12	4/15/39	0	0	0	0	R	0	+	1 year ? 4/38	+	+	+	0
31. W. D. M, W	7	4/25/39	0	0	Fall	7	R	+	+	7 days 4/18/39	+	+	+	0
32. I. T. F, W	5	5/13/39	Pneumonia, empyema, operation	104	0	90	R	0	+	1 day 5/12/39	+	+	+	0
33. L. M. M, W	4	5/15/39	Grippal infection	104	0	35	R	+	+	14 days 5/1/39	+	+	+	0
34. J. R. M, W	2	5/16/39	Grippal infection	103	0	21	R	+	+	14 days 5/2/39	+	+	+	0
35. M. B. M, W	5	5/19/39	Sore throat	103	.. Fall from ladder	3 2	L	+ Left groin	+	3 days 5/16/39	+	+	+	0
36. E. G. M, W	2	5/25/39	0	0	Fall from carriage	7	L	+	+	1 day 5/24/39	+	+	+	0
37. A. P. M, W	5	5/29/39	Sore throat	104	.. Fall from tricycle	14 1	L	Left thigh on awakening in middle of night	+	1 day 5/28/39	+	+	+	0

* Left thigh three-eighths inch smaller than right.

This interpretation of the sequence of events is the direct opposite to that implied by Miller.[8] He stated that although he was uncertain of the original cause, it seemed to him that the epiphysitis was the first manifestation of a pathologic condition of the hip and that it preceded the synovitis or arthritis. On the other hand, the interpretation of Ferguson and Howorth is in agreement with the view expressed by Finder, that the synovitis is the initial stage in most disorders of the hip joint.

Each of the 37 patients whose cases are presented in table 1 was personally examined and selected from children as they appeared in the general pediatric outpatient clinic between January 1937 and June

8. Miller, O. L., in discussion on Ferguson, A. B., and Howorth, M. B.: Coxa Magna: A Condition of the Hip Related to Coxa Plana, J. A. M. A. **104**:808 (March 9) 1935.

Synovitis of the Hip Joint in Children—Continued

Laboratory Data								Course			
					Roentgenograms						
Old Tuberculin Test	Sedimentation Rate of Erythrocytes	Highest Temperature (F.) After First Entry	Kline Test	Blood Count	Date	Interpretation	Follow-Up Roentgenograms and Interpretations	Treatment	Duration of Illness from Onset to Recovery, Days	Present Status	Comments
.....	.....	99	.....	.....	4/17/39	Scoliosis of spine, convex to left: neg. hips	.....	Physical therapy	?	Same	Diagnosis not definite: walks with pelvic obliquity: limitation of motions at right hip unequivocal: under observation
.....	Normal	99.2	Neg.	W.B.C., 7,400: polys. 50%	4/27/39	Neg.	.....	Rest in bed	28	Well	
Neg.	.....	99.2	Doubtful	.....	5/13/39	Neg.	.....	Rest in bed	8	Well	Had pneumonia and empyema January and February 1939: walked well on April 1, 1939: limped May 13, 1939: walked well again after rest in bed
Neg.	Normal	99.4	.....	W.B.C., 6,050: polys. 44%: eos. 0	5/17/39	Neg.	.....	Rest in bed	28	Well	Has vasomotor rhinitis, urticaria, sinusitis and deviated nasal septum: was in bed 4 weeks with upper respiratory infection: walked well 1 week thereafter, then limped
.....	.....	99	.....	.....	.....	.....	.....	Rest in bed	?	?	Heart normal on admission: did not return for follow-up
Neg.	Rapid, 75 mm. in 1 hour	103	Neg.	W.B.C., 13,000: polys. 78%	5/19/39	Neg.	Neg. 5/26/39: Neg. 6/6/39	Rest in bed, extension, sulfanilamide	23	Well	Was put in hospital: observed for possible osteomyelitis of ischium: temperature 101-103 F. for 2 weeks: roentgenograms repeatedly normal: recovered completely
Neg.	.....	99.2	.....	.....	5/25/39	Neg.	.....	Rest in bed	5	Well	
.....	Slightly increased, 26 mm. in 1 hour	99.4	.....	W.B.C., 12,800: polys. 68%	5/29/39	Neg.	.....	Rest in bed	15	Well	

1939. No patient was included in the group unless he satisfied the criteria that are outlined under the definition of terms. The complaints on presentation were of inability or refusal to walk, of limp or of pain in or adjacent to the lower extremity (groin, thigh, knee or lower part of the abdomen). The children were subjected to careful physical examination, both general and local. When no limitation of motion was elicited the patient was treated and observed for some other condition, for example, soft tissue injury or systemic disease, and was not included in the special small group under discussion. If the findings seemed to indicate disease of the hip, further investigation was instituted, the number of studies increasing with knowledge of what to attempt to exclude. It was not always possible to get cooperation in carrying out procedures or in having patients return for follow-up visits. However, in almost every instance a roentgenogram of both hips was obtained

and a series of intradermal tests with old tuberculin in amounts through 5 mg. was given. For the patients seen later, blood counts and erythrocyte sedimentation tests were also done. An orthopedic opinion was sought whenever possible.

It must be confessed that the policy of considering these hip joints as "observation hips" was determined on when a few conditions turned out to be other than transitory synovitis of the hip joint. Among these conditions may be mentioned rheumatic fever, leukemia and lumbar Pott's disease with pain referred to hip and knee and with limitation of motion in the hip. In a few instances the first roentgenograms were interpreted by competent roentgenologists as showing normal hips. Roentgenograms were subsequently retaken and abnormal signs demonstrated. The initial studies were then reviewed and found to have shown early but definite enough deviations from the normal to have warranted their being called pathologic from the beginning. The patients with these roentgenograms, of course, were removed from the group. It should be said in passing that it still is my intention not to be completely satisfied with the diagnosis of transitory synovitis of the hip joint as final but to have the patients restudied at intervals to be certain that insidious and chronic disease of the hip is not being overlooked.

In table 1 the cases are listed with a summary of information on various points.

ANALYSIS OF CASES

History.—This includes color, sex and age of the patient, location and cause of the disorder and season of onset. Of the 37 patients, 35 were white and 2 Negro. The distribution according to sex, 24 male patients and 13 female, agrees with Butler's [2] observations of predominance in the male sex (17 to 5). Finder's [5] figures, however, showed an equal division (11 to 11). The greater incidence among boys, who are more active and reckless than girls, would seem to incriminate injury as a significant etiologic factor. There were reported 45 attacks of synovitis in these 37 children, and table 2 lists the approximate ages at the time of onset:

TABLE 2.—*Children's Ages at Time of Onset of Forty-Five Attacks of Transitory Synovitis*

Age in years	1½	2	3	4	5	6	7	8	9	10	11	12	Total No. of Attacks
Number of children	1	4	0	3	9	3	8	5	3	5	3	1	45

From this table it is clear that the most common period was that between 4 and 10 years of age, during which 36 attacks, or 80 per cent of the total, occurred. The average age of onset was 6.7 years. The average ages for Butler's [2] and Finder's [5] groups were 7 years and 5.4 years, respectively. The youngest child in whose case the diagnosis of transi-

tory synovitis was made was 16 months of age, and the oldest, 12 years. Table 3 classifies the attacks with reference to the extremity involved; multiple attacks and recurrences are listed in sequence: The right hip was involved 26 times and the left 19. There was a recurrence in 1 of Finder's [5] patients, but no report of more than one attack

TABLE 3.—*Distribution of Attacks of Transitory Synovitis with Reference to the Extremity Involved and to the Number of Attacks*

	Children	Hips	Attacks
Single attack, left hip (L)	11	1	11
Single attack, right hip (R)	21	1	21
Two attacks, recurrence (LL)	1	2	2
Two attacks, alternating (LR)	1	2	2
Three attacks, alternating (RRL)	1	3	3
Three attacks, alternating (LLR)	1	3	3
Three attacks, alternating (RLL)	1	3	3
Totals	37		45

involving alternate extremities is available. In no instance in my series were both extremities involved at the same time, and the shortest interval between multiple attacks was nearly two months. However, in the case of recurrence in the same extremity this period was only two weeks.

The name "coxitis of uncertain cause" seems more appropriate than any other when the relevant factors preceding the onset of the complaint are listed, as in table 4: No conclusions can be drawn from these

TABLE 4.—*Possible Contributing Factors Preceding Attacks*

Preceding Factor	Number of Attacks of Synovitis
Common cold	4
Grippal infection or sore throat	5
Pneumonia with pleursy or empyema	2
Purulent otitis media	1
Measles and sore throat	1
Measles, tonsillectomy and injury	1
Sore throat and injury	2
Injury	10
None	19
Total	45

figures, but as would be expected from the predominance of the anomaly in the male sex and as will be considered later in the discussion of the seasonal variation, injury seems to overshadow infection as a possible etiologic factor. This observation is in contrast to the opinions expressed by Butler [2] and Finder.[5] Belmonte [1] mentioned the toxic-chemical, static-traumatic, infectional and climatologic theories of origin. In naming sore throat and even a forced walk as antedating the

anomaly in 2 of his cases, he implied acceptance of infection and trauma as factors. He condemned the theory of some German writers who linked the condition with growing pains in the calves and knees as being caused by benign emboli. Todd [3] also spoke of the traumatic group, or "contusions of the hip," and of the infective forms secondary to febrile conditions. While Finder [5] agreed with Butler [2] in saying that an underlying nonspecific process of infection is responsible, he was unwilling to dismiss entirely other factors that might be at work, namely, trauma, muscular strain and the exanthems.

It was thought that interesting figures could be obtained by arranging the attacks with respect to month and season of onset (tables 5 and 6).

TABLE 5.—*Monthly Distribution of Onset of Attacks of Transitory Synovitis of the Hip Joint*

Month	Number of Attacks	Month	Number of Attacks
January	2	July	5
February	3	August	6
March	4	September	1
April	6	October	5
May	5	November	0
June	0	December	0
		Undetermined	4

TABLE 6.—*Seasonal Incidence of Attacks of Synovitis*

	Spring (May 21 to June 20)	Summer (June 21 to Sept. 20)	Autumn (Sept. 21 to Dec. 20)	Winter (Dec. 21 to May 20)	Undetermined
Number of attacks	16	12	5	8	4
	28		13		

The figures indicating seasonal incidence are used to offset the statistical defect resulting from the incompleteness of the observation period. That period covered, roughly, two and a half years, beginning with January and ending with June; the spring and winter were therefore represented three times and summer and autumn twice. It is permissible, then, to compare the combined incidence of spring and summer with that of autumn and winter. There were 28 attacks in the five periods of spring plus summer as against 13 attacks in the same number of periods in autumn and winter combined, with the time of onset of 4 attacks being undetermined. While the figures are too small to be of statistical significance, the tendency to predominance in spring and summer may be more than a coincidence.

Physical Examination.—Analysis of complaints revealed that pain was present in 38 attacks and absent in 7. The site of pain was most

commonly the thigh or the groin, less often one or the other side of the lower abdominal quadrant or the knee. In most instances the onset was gradual and the pain not severe. On two occasions, however, it was intense enough to awaken the patients during the night and necessitate their being carried to the clinic. In another instance the pain was intermittent. Some children were unwilling to walk and had to be coaxed. A limp was present in 43 instances and absent in 2.

In table 7 are listed the numbers of days between the onset of the complaint referable to the hip and the first entry to the clinic for medical advice.

TABLE 7.—*Intervals Between Onset of Attack and Entry to the Clinic*

Interval	1 Day	2 Days	3 Days	4 Days	5 Days	6 Days	7-14 Days	15-30 Days	31-60 Days	61-90 Days	91-180 Days	1 Yr.	Undetermined
Number of attacks	8	7	5	1	1	0	12	0	2	3	1	1	4

From the table it is plain that most patients came to the clinic after symptoms had persisted one to three days or between one and two weeks. The average for the group, omitting the single case in which the interval was one year, was about twelve days. In Finder's [5] series this average was slightly less than one month.

Limitation of motion in the hip joint is summarized in table 8.

TABLE 8.—*Incidence of Limitation of Motion in the Hip Joint*

		Present	Absent	Undetermined
Resistance to	Abduction	44	0	1
	Extension	38	6	1
	Internal rotation	43	1	1

Muscular atrophy was present in only 1 instance (case 28), in which the affected thigh was ⅜ inch (0.95 cm.) less in circumference than its fellow.

Laboratory Data.—The tuberculin test (intradermal, with amounts of old tuberculin through 5 mg.) was done on 25 of the 37 children. The reaction was positive in 3 patients (cases 4, 15 and 24) and negative in 22.

Roentgenograms of the hips of 31 of the 37 patients were taken in 35 attacks. Six children received no roentgen examinations at any time. In no instance were significant pathologic signs with reference to the hip joint or its contiguous structures detected in the roentgenograms.

Studies of erythrocyte sedimentation were done on 14 of the 37 children. A 20 mm. fall in one hour being considered as the upper limit of normal, results were normal in 9 instances and abnormal in 5.

In 3 of the latter the rates were slightly increased (25, 26 and 35 mm. in one hour in cases 19, 37 and 18, respectively), and in 2 they were fairly rapid (75 and 88 mm. in one hour in cases 35 and 27, respectively).

Blood counts were determined in only 6 instances, being normal in 3 and showing a slight leukocytosis (11,000 to 13,000 white blood cells) in 3 others.

Kline tests were done in only 8 cases, being negative in 7 and doubtful in 1.

Highest temperature readings during the periods of observation after the first entry to the clinic were recorded and found to be below 100 F. in 33 instances and at this level or above in 11. In 2 of the latter the temperature readings ranged between 101 and 103 F., in 1 for three days and in 1 for fourteen days.

Treatment and Course.—Table 9 roughly outlines the therapy.

TABLE 9.—*Methods of Treatment of Transitory Synovitis*

Therapy	Number of Attacks
Rest in bed with or without salicylates	37
Rest in bed, extension and sulfanilamide	1
Rest in bed, Buck's extension and cast	1
Rest in bed and supinating heel braces	1
Rest in bed followed by physical therapy	3
Physical therapy alone	1
None	1

The course was favorable in all except 6 of the patients. In 5 of the latter the outcome was undetermined because of failure of cooperation in follow-up visits. The other patient has shown some improvement but not complete resolution as yet.

TABLE 10.—*Total Duration of Illness from Onset to Termination*

	Less							Undeter-
Duration, days	than 7	7-14	15-21	22-28	29-60	61-90	90+	mined
Number of attacks	2	10	7	4	5	2	3	12

The durations of the complete course, from the onset of the chief complaint to the disappearance of symptoms and signs, is tabulated in table 10.

Most of the children recovered in from seven to sixty days after onset, the average for the group being thirty-two days. This duration approximates the reports of other authors.[9] The large number of undetermined periods occurred in cases of attacks either of indefinite onset or of unknown terminal date or both.

9. Belmonte.[1] Butler.[2] Finder.[5]

COMMENT

It must be emphasized that the term transitory synovitis of the hip joint is one of convenience and that the diagnosis can be arrived at only by exclusion after prolonged observation. The site and the underlying pathologic condition furnish enough evidence to justify one's regarding the anomaly as synovitis, and when the process is characterized by subsidence in a relatively short time, as transitory synovitis. In the selection of the 37 cases an attempt was made to be wholly objective. Only those children with symptoms and signs for which no obvious explanation could be found were included. The children came to the clinic with a complaint of pain referable to the lower extremity, of limp or of unwillingness to walk. A history was taken pertaining to preceding factors possibly significant, and including a record of the intervals between the appearance of those factors and the onset of the chief complaint, as outlined in table 1. A complete physical examination was done to find, if possible, a systemic condition of which the articular or muscular pain might be part (for example, rheumatic fever, purpura or Still's disease). A special local examination was then made. The child was undressed completely and placed on a flat, broad-topped wooden table. With gentleness in manipulation as a cardinal requisite, maneuvers were begun, first with the sound limb, to gain the patient's confidence and to allow comparison with the affected extremity. The procedure followed was, with slight variation, essentially as outlined by Albee.[10]

The normal movements of the hip joint are flexion, extension, abduction, rotation and circumduction. When these were carefully done, it was extension, abduction and internal rotation that showed the greatest deviation from the normal in the cases under consideration. Because of the importance of these maneuvers a detailed description of them seems indicated:

Abduction and internal rotation are investigated with the patient in the supine position. For the performing of internal rotation, while one extremity is completely extended and untouched the other is flexed at the hip until the thigh is perpendicular to the body; then, with the knee also flexed to a right angle and fixed by the left hand of the examiner, the ankle is grasped by the examiner's right hand and moved directly outward. The movement is in effect an internal rotation of the femoral head. Pain is usually evinced when this is tried on the involved extremity.

Abduction is carried out in a slightly different way. Flexion of thigh and knee are performed as for the preceding movement, as a rule eliciting no resistance. Then, with the right extremity being tested, the examiner's left hand is placed on the patient's right knee and his right hand under the left buttock. The right extremity is then gently and slowly rotated in a smooth arc outward and downward toward the table. Normally the left side of the body moves little if at all. When

10. Albee, F. H.: Injuries and Diseases of the Hip, New York, Paul B. Hoeber, Inc., 1937.

there is spasm of the adductor muscles of the hip on this tested right side, the maneuver brings the following result: The left side of the pelvis accompanies the movement of the right, and the left buttock is lifted off the examiner's right palm on the table. The height of the elevation may be observed by sighting the table top. Comparison of this movement with that for the sound limb is important in detecting slight degrees of muscular spasm.

Extension is done with the patient in the prone position. In a test of the right extremity, the palm of the examiner's left hand is placed lightly on the patient's right buttock. With the patient's thigh kept flat on the table the knee is flexed to a right angle. The examiner, holding the ankle lightly with his right hand, gently but fairly quickly raises and lowers the flexed leg several times. Even minimal spasm is detected by the hand on the buttock of the involved extremity. In the sound limb, however, there is no resistance to a similar maneuver. This is especially striking in alternate comparisons between the two sides. In the testing of the left hip, the examiner's hands may be reversed, the left being kept on the patient's left buttock and the right being used at the left ankle to raise the leg.

As soon as one uninitiated is shown these maneuvers and is allowed to carry them out for himself, he is able convincingly to detect the abnormal conditions when they exist. Palpation for fulness and for points of tenderness, tape measurements of corresponding areas on both extremities for evidences of shortening or of muscular atrophy and inspection of gait are among the other procedures that should be carried out in a routine examination of the hip. Other tests that have proved helpful in the hands of the individual examiner should not be neglected.

Once the diagnosis of disease of the hip was made in my series it became important to determine the nature of the condition, its site and its prognosis. Laboratory aids, already mentioned, were important in the consideration of differential diagnosis. These included a series of intradermal tuberculin tests, in several instances a white blood cell and a differential count, an erythrocyte sedimentation test and a roentgenogram of the pelvis taken so as to include both hips. In special instances lateral roentgenograms were also taken.

Great care and close study are needed in the interpretation of the roentgenogram of the hip. In the past the pathologic conditions in most cases of disease of the hip joint were so far advanced that when the roentgenograms were taken the diagnosis was obvious to any one with even a slight knowledge of roentgenology. The goal to be striven for is the recognition of initial changes such as the early abnormal conditions of bone texture and joint outline. For this the use of a magnifying hand lens and a metric rule is invaluable.

In connection with their studies of coxa plana Ferguson and Howorth [7] listed in a comprehensive outline the important roentgenographic features of that disorder from the incipient stage progressively through the latest phases. However, the roentgenogram should even facilitate an early decision as to whether and when an "observation hip"

regarded up to then as a hip joint with transitory synovitis should be excluded from that convenient classification. Among the early roentgenographic changes in the incipient stage of coxa plana are listed the following conditions: distention of the capsule, widening of the cartilaginous joint space, slight flattening of the crest of the head, slight radiolucency of the neck at the epiphysial disk and widening of the epiphysial disk. In the same paper are also set down the early roentgenographic changes of tuberculous coxitis. These include thinning of the joint space, local decalcification, lack of broadening of the head and neck and occurring, somewhat later, destruction at the surface of the bone.

These—coxa plana and tuberculous coxitis—are only two important conditions which, early in their course, may be confused, with transitory synovitis of the hip joint. As a matter of fact, it is advisable to be able to detect not only the full-blown signs of all roentgenographically abnormal hip joints but their earliest manifestations as well, before expressing judgment of a roentgenogram as unequivocally negative.

The diagnosis of tuberculous coxitis plagued many early observers. A series of negative reactions to tuberculin rules out this diagnosis. In an instance of a positive test, repeated roentgenographic studies over a long period of time may be necessary before one can decide. As Butler [2] has suggested, the chances of the roentgenogram's being negative in the early stage of tuberculosis of the hip are slight. Belmonte [1] pointed out the occurrence of atrophy of the muscles of the buttock or thigh early in the course of tuberculous coxitis as against absence of this anomaly in transitory synovitis of the hip joint. Todd [3] said that he considered the best test to be the effect of mobilization followed by release, by allowing the patient to run about for a week: Tuberculous coxitis recurs; transitory synovitis does not. I am not in agreement with these points of view, since in case 28 of the current series both recurrence and atrophy were present and the tuberculin test was negative. Laboratory tests are less fallible than clinical impressions and merit preference in the forming of final opinions. When other means have proved of no help, aspiration of the joint and inoculation of a guinea pig with the synovial fluid may be resorted to. Finally, recourse may be had to open operation and to biopsy of synovia and adjacent structures.

Of the single, easily accomplished and reliable procedures, roentgen examination remains the most helpful in the separating of transitory synovitis from other conditions of the hip. The list of such conditions, moreover, is by no means an inconsiderable one. For example, Albee,[10] in his discussion of the differential diagnosis of tuberculous coxitis, has arranged a table of 22 pathologic conditions, giving points of resem-

blance and difference. Since the same list may be used for transitory synovitis of the hip joint it is given in abbreviated form here, with points of similarity and difference omitted.

Conditions Resembling Transitory Synovitis of the Hip Joint

1. Local irritation (vaginitis)
2. Acute adenitis
3. Local injury
4. Anterior poliomyelitis
5. Acute arthritis and epiphysitis
6. Rheumatism
7. Lumbar Pott's disease
8. Knee joint disease
9. Coxa vara
10. Hysterical joint
11. Periarticular disease
12. Perinephritis and appendicitis
13. "Growing pains"
14. Scurvy
15. Arthritis deformans of hip
16. Atrophic polyarthritis
17. Sacroiliac disease
18. Pelvic disease
19. Disease of bursae about joint
20. Fracture of neck of femur
21. Epiphysial fracture
22. Congenital dislocation of hip

To these may be added, among others, without exhausting the conditions that may be confused with transitory synovitis of the hip:

23. Tuberculous coxitis
24. Coxa plana
25. Coxa magna
26. Transient epiphysitis
27. Traumatic chondritis
28. Hemophilia
29. Psoas myositis
30. Coxa valga
31. Syphilitic epiphysitis
32. Neoplasm
33. Sepsis
34. Osteochondritis ischiopubica
35. Intermittent hydrarthrosis
36. Leukemia
37. Purpura
38. Cretinous hip
39. Sickle cell anemia
40. Osteomyelitis of innominate bone

Most of these can be eliminated by a combination of laboratory tests and waiting. One of these conditions merits more than passing comment, namely, intermittent hydrarthrosis. Berger [11] recently reported a case in which this condition involved the knee joint and in which the symptom complex was proved to be of allergic origin. It is not inconceivable that the hip joint could be the site of such a process. Albee,[10] in his discussion of synovitis of the hip joint, spoke of the difficulty of detecting the presence of fluid in that joint. This is so because the deep situation of the hip joint and its large muscular encirclement make it impossible to palpate any distention of the capsule. An intermittent hydrarthrosis, then, could be present in the hip joint without detection except from the painful limited motion and muscular spasm. Todd [3]

TABLE 11.—*Possible Allergic Manifestations in Eight Cases of Transitory Synovitis of the Hip Joint*

Case Number	Attacks of Synovitis	Duration, Days	Allergic Manifestations (History)
1	3 (LLR)	(*a*) ? (*b*) ? (*c*) 16	No manifestation mentioned, but personal history of decided allergy
2	3 (RRL)	(*a*) ? (*b*) 30 (*c*) 21	Asthma; papular urticaria (?)
4	1 (L)	90	Allergic rhinitis, infectious asthma
10	1 (L)	16	Familial allergy; angioneurotic edema, urticaria, abdominal pain (?)
16	1 (R)	8	Familial allergy; intolerance to egg, abdominal pain (?)
23	1 (L)	8	Familial allergy; papular urticaria (?)
28	2 (LL)	(*a*) 23 (*b*) 4	Vasomotor rhinitis (?)
33	1 (R)	28	Vasomotor rhinitis (?); urticaria

may have been thinking of a related process when he spoke of the condition of "intestinal pseudocoxitis." This he described as usually occurring in a constipated child, lasting about two weeks and responding to treatment with laxatives and a nonprotein diet. He indicated that it might be a "kind of protein sensitization." With this attractive theory as a basis one might wish to speculate further on the unusually large number of children (8 out of 37) in the group under observation who had a suggestive personal or familial history of allergy. Data on these 8 children are listed in table 11.

It is noteworthy that in 3 of the 8 cases, repeated attacks of synovitis are recorded, fulfilling the criterion of intermission. Since no blood counts for eosinophilia were done, nor aspiration and studies of the synovial fluids, one could not say with any certainty what the diagnosis

11. Berger, H.: Intermittent Hydrarthrosis with an Allergic Basis, J. A. M. A. **112**:2402 (June 10) 1939.

should have been. Strictly speaking, hydrarthrosis, while transitory, would fall out of the class of synovitides, since it does not conform with the accepted definition of inflammation. In a further study of this group, however, there is one factor that would militate against the diagnosis of an allergic process as a basis, namely, the duration. This ranged from four to ninety days, with an average of twenty-four days in the cases in which the duration was known. This is in striking contrast to the well recognized ephemeral character of all allergic processes (in the narrower meaning) and would tend to throw doubt on such a basis in the present series.

The question of whether the basis of the condition was subacute rheumatic fever involving the hip joint was raised several times and could not be answered immediately. As in the consideration of allergic disease, so too in the consideration of rheumatic fever, distinguishing features were present: The longer duration, the limitation of the pain to one joint and the absence of abnormal cardiac findings were not characteristic of rheumatism.

It is important to be aware of the differential diagnosis of transitory synovitis of the hip joint, because the treatment and prognosis are radically different in the various conditions. As shown in table 10, the duration of transitory synovitis of the hip joint in most children varies from seven to sixty days, requiring an average period of thirty-two days for recovery to ensue. Most of the children were advised to rest in bed for two weeks, but it is by no means certain that the order was carried out for more than a few days. While in addition to the advice to rest in bed, use of Buck's extension, immobilization in plaster of paris hip spica, and administration of salicylates and sulfanilamide, it seemed advisable to employ in accordance with the rapidity of response to the treatment, first, physical therapy, and then elimination of foci of infection and general hygienic measures, still in most instances simple rest in bed for a week or two was adequate alone to effect a satisfactory outcome, at least for the time of observation. This favorable termination suggests the interdiction of all but conservative measures in the treatment of these children but does not obviate the necessity for keeping them under prolonged observation, limiting their activity and making frequent roentgenographic examinations. It must be admitted that in the present series the studies and management fall far short of the ideal. As already implied, an ideal program for dealing with any child who enters with a complaint referable to the lower extremity would, when hip joint disease is suspected, include the following measures:

1. The taking of a complete history.
2. A complete general and a special local examination.

3. Laboratory studies, that is, a complete blood count, erythrocyte sedimentation test, tuberculin series, Kline (or related) study and roentgenograms of both hips.

4. Injunction to rest in bed for two weeks.

5. Follow-up examinations and roentgen examinations at the end of one month, six months, twelve months and yearly thereafter for three years.

SUMMARY

1. The condition known as transitory synovitis of the hip joint in children (a form of "observation hip") is described.

2. Closely related anomalies of the hip joint previously reported are reviewed and their pathologic and roentgenographic differences stressed.

3. Thirty-seven cases of transitory synovitis of the hip joint in children observed for two and one-half years are presented and analyzed.

4. The technic of an adequate examination of the hip joint in this condition is detailed.

5. The importance of roentgenographic study and interpretation and of other laboratory aids in the differential diagnosis is pointed out.

6. The question of the relationship of allergy to transitory synovitis of the hip joint is raised.

7. A program of management of any child with complaints referable to the lower extremity is suggested.

DURATION OF IMMUNITY TO DIPHTHERIA ACHIEVED BY TOXIN-ANTITOXIN AND ALUM-PRECIPITATED TOXOID

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In the year 1932 the use of alum-precipitated toxoid began to supplant the use of toxin-antitoxin and fluid toxoid for immunization against diphtheria. The simplicity of the single injection, its freedom from reactions in young children and the rapid development of immunity appealed to physicians and public health officials. It was assumed that the duration of the immunity was the same as that obtained by toxin-antitoxin, which Park and his co-workers had found to be ten years or more in 80 per cent of cases.[1] Recently, however, several reports have appeared in the literature which suggest that the persistence of immunity (as determined by a negative Schick test) produced by alum-precipitated toxoid is not so long as was previously believed, although there are also other reports indicating the persistence of a high degree of immunity. A search of the literature has not revealed any studies covering over three years. We feel that we are the first to report comparative results of Schick tests on groups of school children *five* years after immunization with toxin-antitoxin and with alum-precipitated toxoid.

RESULTS OF OTHER STUDIES

In a study under the direction of Deputy Health Commissioner W. H. Best of New York city,[2] it was learned that in 668 cases of diphtheria occurring in Manhattan and Brooklyn in 1936 in children under 15 years of age, 249 patients were children who had received immunizing injections. (It was not stated that these children had received the Schick test and had reacted negatively after the injections.) When the 249 cases were tabulated according to the time between the preventive inoculation and the development of diphtheria, it was found that the number of cases increased with the length of the interval. For instance, 22 cases of the 249 occurred in three to twelve months, while 59 cases occurred after twenty-four to thirty-six months and 58 after thirty-six to forty-eight months. There is no doubt that the children

1. Park, W. H.: Duration of Immunity Against Diphtheria, J. A. M. A. **109**:1681 (Nov. 20) 1937.

2. Increase in Diphtheria Prevalence, Quart. Bull. New York Dept. Health **5**:8 (Feb.) 1937.

afflicted lost their immunity, if indeed they had ever had any conferred by their previous injections! A similar study made in 1937 fully confirmed the results obtained in 1936.[3]

Fraser and Halpern, of Toronto,[4] studied the duration of immunity obtained after use of alum-precipitated toxoid compared with that achieved by injection of fluid toxoid; the accurate method of determining the antitoxin content of the blood of immunized children was used. They gave 40 children 1 cc. of alum-precipitated toxoid and 35 children three 1 cc. doses of plain toxoid. In ten weeks only 62 per cent of the children who had received the single dose of alum-precipitated toxoid had more than $\frac{1}{100}$ unit of antitoxin per cubic centimeter of blood, as compared with 91 per cent of the group who had received fluid toxoid ($\frac{1}{100}$ unit of antitoxin per cubic centimeter of blood gives a negative Schick test). After twelve months the figures were 19 per cent and 91 per cent respectively, showing a rapid loss of immunity when the alum-precipitated toxoid was used.

Park [1] reported a similar study by Blum, of the City of New York Department of Health, except that the immunity was determined by the Schick test. The following table shows the results clearly:

TABLE 1.—*Duration of Diphtheria Immunity Produced by Various Methods in Children Whose Initial Reaction to the Schick Test Were Positive*

	Interval (Yr.) Between Immunization and Schick Test	Total	Number of Positive Schick Reactions	Percentage of Positive Schick Reactions	Percentage of Negative Schick Reactions
3 × 1 cc. toxin-antitoxin.....	1-2	39	3	8	92
	2-3	72	3	4	96
2 × 1 cc. fluid toxoid.....	1-2	66	5	7	93
	2-3	20	0	0	100
1 × 1 cc. alum-precipitated toxoid	1-2	135	54	40	60
	2-3	63	52	82	18

Certainly a procedure that produces only 18 per cent immunity after two to three years stands in need of revision.

Reports much more favorable to the use of alum-precipitated toxoid have been made by Farago,[5] Dean and Hyman [6] and Bundesen, Fishbein and White.[7]

3. Desirable Procedure for Diphtheria Immunization, Quart. Bull. New York Dept. Health **6**:56 (May) 1938.

4. Fraser, D. T., and Halpern, K. C.: Diphtheria Toxoid: Comparison of One Dose Alum-Precipitated with Three Doses of Unmodified Toxoid, Canad. Pub. Health J. **26**:469 (Oct.) 1935.

5. Farago, F.: Immunization Against Diphtheria: Relative Value of Two Methods as Shown by Schick Testing, Am. J. Hyg. **22**:495 (Nov.) 1935.

6. Dean, A. S., and Hyman, S.: Alum-Precipitated Toxoid for Prevention of Diphtheria, Am. J. Pub. Health **28**:1170 (Oct.) 1938.

7. Bundesen, H. N.; Fishbein, W. I., and White, J. L.: Diphtheria Immunity in Chicago, J. A. M. A. **112**:1919 (May 13) 1939.

In 1935 Farago published the results of a study made on large groups of school children in Hungary. Three months after immunization with 1 cc. of alum-precipitated toxoid, 93.3 per cent of 2,652 children were found to have negative Schick reactions, while after twelve months a slightly greater number, 93.7 per cent, had negative reactions. Farago has no figures available beyond the twelve month period.

Dean and Hyman gave the Schick test to their patients four, sixteen and twenty-eight months after immunization with 1 cc. of alum-precipitated toxoid. They divided their patients into three groups according to the results: (1) 302 children who had positive Schick reactions and who had not been previously treated showed after four, sixteen and twenty-eight months 86 per cent, 87 per cent and 89 per cent immunity respectively, indicating an actual slight increase in immunity with the passage of time; (2) 140 children who had positive Schick reactions in spite of previous treatment showed 94, 90 and 98 per cent immunity after four, sixteen and twenty-eighth months respectively, and (3) 178 children who had been given neither the Schick test nor treatment previous to immunization by Dean and Hyman showed 98, 98 and 95 per cent immunity after the same intervals as in the previous two groups. The authors concluded, therefore, that twenty-eight months after treatment with 1 cc. of alum-precipitated toxoid at least 89 per cent of children would have a negative Schick reaction.

These results are substantiated by the work of Kereszturi [1] who used alum-precipitated toxoid prepared by the New York city department of health in a small study at the Medical Center of Columbia University. Two groups of 21 children each were treated, one with three doses of toxin-antitoxin and one with 1 cc. of alum-precipitated toxoid. Thirty-nine months later 95 per cent of the first group gave negative Schick reactions, and after twenty-seven months exactly the same percentage of the second group likewise gave negative reactions—a highly satisfactory result.

Bundesen, Fishbein and White [7] have recently reported a comprehensive survey of diphtheria immunity in Chicago. They determined the degree of immunity by the antitoxic titer of the blood as well as by the Schick test in groups of children who had previously been immunized by different methods at varying intervals. The greatest interval was in the group of 125 children who had received three doses of toxin-antitoxin in 1930. In 1936 only 2 of these children had positive Schick tests; i. e., a 98.4 per cent rate of immunity existed for this group six years after treatment. Unfortunately the time interval for the group who received one dose of alum-precipitated toxoid was only two years, next to the shortest of all the periods. In this group, 113, or 95 per cent, of 119, had negative Schick tests, certainly a worth while if not a 100 per cent result. Bundesen, Fishbein and White could make

an even finer contribution to this rather confused subject by carrying on their studies with the same children until five years or even more has elapsed in all their groups since the time of the original inoculations, and it is sincerely to be hoped that they will be able to do so.

STUDIES IN EAST ORANGE

For sixteen years group immunization of school children against diphtheria has been carried out in the East Orange public schools. Three doses of antitoxin were last used in the fall of 1932, and a 1 cc. dose of alum-precipitated toxoid prepared by the New York city department of health was used first in the fall of 1933 and has been used subsequently. Hence in this study the same material was employed as in Blum's and Kereszturi's studies.

TABLE 2.—*Duration of Diphtheria Immunity After Five Years Produced by Two Different Methods in East Orange Public School Children* *

	Number of Children Treated	Number of Negative Schick Reactions After 12 Mo.	Number of Negative Schick Reactions After 5 Yr.	Percentage with Negative Schick Reactions After 5 Yr.
3 × 1 cc. toxin-antitoxin	78	78	75	96
1 × 1 cc. alum-precipitated toxoid	72	72	58	80

* All the children with positive Schick reactions were immediately given injections of 1 cc. of alum-precipitated toxoid.

Seventy-eight children were found in the fall of 1937 who had been treated with three 1 cc. doses of diphtheria toxin-antitoxin in 1932 and *who were known to have negative Schick reactions one year later.* They were again given the Schick test five years after their original treatment, and 75 children, or 96 per cent, still reacted negatively.

The more important group, the group immunized with 1 cc. of alum-precipitated toxoid in the fall of 1933, comprised 72 children. They were all tested and *known to have negative Schick reactions in 1934.* In the fall of 1938 they were again given the Schick test, and only 58 children, or 80 per cent, still reacted negatively; thus, as determined by the Schick test, a reversion from immunity to nonimmunity in four years in 20 per cent of cases was indicated. It appears, then, that while alum-precipitated toxoid produces a negative Schick reaction promptly in a high percentage of cases, the result is transient and disappears after five years in about 20 per cent of children.

East Orange has a school population of nearly 10,000, yet much difficulty was met in finding these groups of 78 and 72 children respectively, partly because of the transient type of population living in that city and partly because of the large number of children under the care

of private physicians. It is probably the shifting of the type of population encountered in clinics which explains in large measure the great scarcity of reports on the duration of artificial immunity beyond three years.

The results of this study bear out the general belief that artificial immunity to diphtheria as obtained from a single dose of 1 cc. of alum-precipitated toxoid does not last so long as that obtained from the use of the older preparations. It is felt that the practical application of this knowledge consists not in a return to the use of the older toxin-antitoxin preparations, but in the use of two 1 cc. doses of alum-precipitated toxoid two or three weeks apart and in the readministration of the Schick test to children at regular intervals after the original injections. Since the ideal time for immunization is around six to nine months, Schick tests should be done at the ages of 12 months, 3 years and 5 years, or just before entering school. In the event that the reaction becomes positive, another cubic centimeter of toxoid should be given with subsequent retesting.

SUMMARY AND CONCLUSIONS

The great scarcity of reports on duration of diphtheria immunity beyond three years is pointed out. The report of a study covering a five year interval is presented:

A group of 72 school children, who were immunized with 1 cc. of alum-precipitated toxoid and were found to have negative Schick reactions twelve months later, were again given the Schick test five years after the original injection. Only 58 children, or 80 per cent, still reacted negatively.

This group was compared with a group of 78 which was exactly similar except that the children of the second group had been treated with three 1 cc. doses of toxin-antitoxin. Five years later there were in the second group 75 children, or 96 per cent, who still gave negative reactions.

The following conclusion was reached: One cubic centimeter of alum-precipitated toxoid is not entirely satisfactory for immunization against diphtheria.

A suitable program of immunization and testing is suggested.

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Case Reports

PERIARTERITIS NODOSA COMPLICATING SCARLET FEVER

WITH AN UNUSUAL SYNDROME OF NEPHRITIS AND POLYNEURITIS

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Periarteritis nodosa occurring during the course of scarlet fever with the syndrome of nephritis and polyneuritis has not been encountered in a survey of the literature. Vascular lesions, arterial or venous, are not commonly observed, and reports of such complications are sparse. Cases of acute obliterative arteritis occurring in the second to third week of scarlet fever and complicated with gangrene of the lower extremities have been described by Pearson and Littlewood,[1] Bindschedler, Tassovatz and Flamée [2] and Anghelesco.[3] Rothstein and Welt [4] reported an interesting case in which cervical adenitis and abdominal pain developed in a 7 year old boy in the third week of scarlet fever; pain in the foot, thumb, groin and scrotum occurred later (fourth, fifth and sixth week), followed by a gradual onset of pallor, weakness, cyanosis and edema and finally terminal cardiac decompensation. At necropsy periarteritis nodosa was found complicated with rheumatic endocarditis. At a recent clinical conference at the Willard Parker Hospital,[5] New York, there was reported the occurrence of scarlet fever, periarteritis nodosa, aneurysm of the coronary artery with spontaneous

From the Municipal Contagious Disease Hospital.

Read before the Chicago Pediatric Society, Nov. 21, 1939.

1. Pearson, A. E., and Littlewood, H.: Case of Dry Gangrene of Both Lower Extremities Complicating Ordinary Scarlet Fever, Lancet **2**:84, 1897.

2. Bindschedler, J. J.; Tassovatz, B., and Flamée, A.: Oblitération artérielle au cours d'une scarletine, Strasbourg méd. **90**:810 (Dec. 25) 1930.

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rupture and hemopericardium in a 5 year old girl. Autopsy did not reveal any significant changes in any other organs. The case to be reported here is unusual because of the syndrome of nephritis and polyneuritis.

REPORT OF CASE

History.—An 8 year old boy was admitted to the Municipal Contagious Disease Hospital on Sept. 19, 1938, with scarlet fever of four days' duration. He had been given 20 cc. of scarlet fever convalescent serum just prior to admission.

On admission the patient appeared comfortable, not dyspneic or cyanotic. Circumoral pallor was present, and there was a faint punctate eruption over the trunk. The temperature was 100.4 F., the pulse rate 96 and the respiratory rate 24 per minute. There was nothing unusual about the eyes, ears or nose. The tongue was red anteriorly and heavily coated posteriorly; the papillae were prominent. Cervical and inguinal lymph nodes were palpable. The chest and heart were normal. The abdomen was soft, and no masses were felt. The extremities were not unusual. Neurologic examination gave negative results. The white cell count on the patient's admission was 12,200 per cubic millimeter. The urine was normal. The Schick test gave negative results.

Course in the Hospital.—The course in the hospital was uneventful until October 4, when the patient vomited and the temperature rose to 103 F. The following day the cervical lymph nodes became enlarged and tender; the urine showed a trace of albumin and a few pus cells. On October 7, twenty-two days after the onset of the disease, the urine showed many red blood cells; a diagnosis of acute glomerulonephritis complicating scarlet fever was considered. From October 8 to 13 the urine showed increasing amounts of blood and albumin; the temperature varied from normal to 102 F., and the blood pressure was about 120 mm. of mercury systolic and 80 mm. diastolic. The urinary output was well maintained, and there was no peripheral edema. The patient was kept on the ward diet, which included moderate amounts of protein and a low intake of salt. Cod liver oil and orange juice were added daily. The total daily intake of fluid averaged 1,200 cc.

On October 13 the blood pressure rose to 160 mm. systolic and 120 mm. diastolic. There was no headache, nausea or vomiting. The patient was given magnesium sulfate both in 50 per cent solution by mouth and in 25 per cent solution intramuscularly twice daily. Two days later the blood pressure dropped to 140 mm. systolic and 90 mm. diastolic; the urine showed granular and hyaline casts in addition to blood and albumin. Results of chemical studies of the blood were as follows: nonprotein nitrogen, 68.2 mg. per hundred cubic centimeters; urea, 45.1 mg.; uric acid, 8 mg.; creatinine, 1.8 mg., and sugar, 81 mg.

On October 17 the temperature rose to 104 F., and the patient complained of headache. His face appeared puffy, and there was marked tenderness over the region of both kidneys. The blood pressure was 150 mm. systolic and 90 mm. diastolic. Tests for concentration of the urine showed a fixation of the specific gravity at 1.010. The urine continued to show increasing amounts of albumin, red cells and granular and hyaline casts.

On October 19 the patient complained of extreme fatigue and double vision. Examination of the fundi revealed nothing unusual, and there were no abnormal neurologic findings. The hemoglobin content was 68 per cent (Dare), the red cell count 4,370,000 and the white cell count 20,950 per cubic millimeter. On October 21 headache and abdominal pain occurred. There was some fulness of the abdomen but no rigidity or spasticity. Exquisite tenderness was elicited over the region of both kidneys.

On October 24 nausea and vomiting recurred, and there was a urinous odor to the breath. The blood pressure varied from 130 to 150 systolic and from 90 to 110 diastolic. The liver was palpable 4 cm. below the costal margin in the right midclavicular line. The hemoglobin content was 80 per cent (Dare), the red cell count 3,750,000 and the white cell count 17,880 per cubic millimeter. The blood chemistry indicated further retention: nonprotein nitrogen, 105.9 mg. per hundred cubic centimeters; urea, 57.1 mg.; creatinine, 2.7 mg.; cholesterol, 128 mg., and total protein, 6.1 mg. (albumin 2.8 mg. and globulin 3.3 mg.). During the course of the next few days the patient suffered from anorexia, drowsiness, intermittent headache, vomiting and abdominal pain. The temperature ranged between 100 and 102 F.

On October 29, forty days after the onset of the disease, the patient complained of vague shooting pains in the left arm. Examination revealed nothing unusual. The blood pressure ranged about 130 mm. systolic and 85 mm. diastolic. The blood chemistry indicated some improvement: nonprotein nitrogen, 81.1 mg. per hundred cubic centimeters; urea, 16.5 mg.; creatinine, 2 mg., and cholesterol, 113 mg.

On November 5 the patient complained of pain in the right elbow and weakness in both arms. Neurologic examination revealed the following changes: The right upper extremity showed weakness of flexion and extension at the elbow, loss of flexion and weakness of extension at the wrist, poor abduction and adduction of the fingers and loss of grip. Sensory examination in the same extremity (light touch and pinprick) revealed loss of sensation along the distribution of the lateral antebrachial cutaneous nerve and complete glove anesthesia. The motor changes within the left upper extremity were similar to those in the right except for wristdrop and weakness of flexion at the wrist. The sensory changes consisted of hyperesthesia along the distribution of the medial brachial and antebrachial cutaneous nerves and along the hypothenar region and of anesthesia along the lateral and antebrachial cutaneous nerves and medial half of the palm. There was muscular tenderness in both extremities. There were no changes within the shoulder girdle or muscles of the trunk. The cranial nerves were intact. The reflexes were diminished. Acute polyneuritis was considered.

On November 8 there was hyperesthesia of the thenar eminence of the right hand with return of sensation of the proximal portion of the dorsum of the hand. The left lower extremity showed weakness of flexion and extension at the knee and some diminution of sensation along the medial aspect of the leg. The muscles of the calf were markedly tender. Examination of the blood revealed a hemoglobin content of 45 per cent (Dare) and a red cell count of 3,120,000 per cubic millimeter. The patient was given 360 cc. of citrated blood intravenously. On November 9 chemical analysis of the blood showed further improvement: nonprotein nitrogen, 51 mg. per hundred cubic centimeters; urea, 31.5 mg.; uric acid, 3.3 mg., and creatinine, 1.7 mg.

On November 11 the right upper extremity showed some return of flexion and extension at the elbow, and the left upper extremity showed some return of sensation over the distribution of the lateral antebrachial cutaneous nerve. The left lower extremity was almost normal.

On November 13 lumbar tap revealed a normal fluid. The reactions to the Wassermann test and the colloidal gold test were normal. During this period the urinary output was well maintained. The patient consumed a fair amount of a high vitamin, high calory diet, reenforced with cod liver oil and orange juice. In addition he was given iron and ammonium citrates U.S.P. and tablets of brewers' yeast.

On November 23 the urinary output was poor, and the patient appeared drowsy and listless. The blood pressure was maintained at about 150 mm. systolic and 120 mm. diastolic. The eyes were puffy. There was a urinous odor to the breath, and the tongue was dry and furred. The chest was clear. The heart was enlarged to the left, and there was a soft systolic murmur over the apex. The liver and spleen were not palpable. Both arms showed some return of motor power. Examination of the right upper extremity revealed normal sensation over the distribution of the lateral antebrachial cutaneous nerve and hyperesthesia over the medial half of the palm and the entire dorsum of the hand. The left upper extremity showed further improvement of sensation over the distribution of the lateral antebrachial cutaneous nerve.

On November 25 chemical analysis of the blood revealed: nonprotein nitrogen, 40.7 mg. per hundred cubic centimeters; urea, 17.2 mg.; uric acid, 3.7 mg.; creatinine, 1.6 mg., and cholesterol, 145.5 mg. The patient was given 250 cc. of citrated blood intravenously.

On November 29 the respirations were 60 per minute, and there was a marked urinous odor to the breath. The patient was drowsy and stuporous at times. There were projectile vomiting and poor urinary output. The patient complained of headache and abdominal pain. Examination of the fundi revealed fulness of the retinal vessels but no hemorrhages or exudates. The cardiac dulness was increased to the left and to the right, and the systolic murmur was louder and more prolonged. The liver extended 3 cm. below the costal border and was exquisitely tender. The spleen was not palpable. There were no other changes in the extremities. The reflexes were decidedly diminished. Another transfusion of 200 cc. of citrated blood was given and was followed by a continuous intravenous injection of 5 per cent dextrose in lactate-Ringer solution. Subsequently marked dyspnea and cyanosis developed, with dilatation of the alae nasi. The temperature ranged between 99 and 102 F. The index and ring fingers of the right hand showed beginning dry gangrene, and the finger nails had fallen off. The blood culture was positive for Streptococcus haemolyticus.

On December 2 many petechiae were noted over the trunk. The temperature ranged between 101 and 102 F. The breath sounds were impaired over the upper part of the left side of the chest and the left axilla, where also crepitant rales were heard. The following day the patient became moribund and had frequent coughing spells, which were nonproductive, and gasping respirations. The temperature ranged from 101 to 105 F. Edema of the face, scrotum and extremities developed. Pneumonic signs appeared in both lungs, and the liver descended to the umbilicus. The abdomen bulged in both flanks, and a fluid wave was elicited. Petechiae were noted in the conjunctivas.

On December 4, the eighty-first day of the disease, the patient died.

Gross Postmortem Examination.[6]—Many petechiae were present over the trunk, and a few were noted in the conjunctivas. The distal portions of the right index and the right ring finger were dry, shrunken and blue-black, and the finger nails were missing. The face, scotum and lower extremities were edematous.

There was about 100 cc. of straw-colored fluid within the peritoneal cavity; the peritoneal surfaces were smooth and glistening. The inferior margin of the liver extended 7 cm. below the costal border in the right midclavicular line and 10 cm. below the xiphoid process. The anterior surface of the liver was covered by a thin layer of light gray fibrin. Each pleural cavity contained about 500 cc.

6. Necropsy was performed by Dr. Victor Levine.

of slightly cloudy fluid with many fibrin flakes; the pleural surfaces were finely granular. The pericardial cavity contained about 50 cc. of clear, straw-colored fluid.

The heart weighed 250 Gm. The pericardium was smooth, and the myocardium was flabby and pale brown-red. The atrioventricular, pulmonic and aortic orifices were normal. The coronary vessels revealed nothing unusual.

Cut sections of the lungs showed recent infarcts in the upper lobe of both lungs and in the middle lobe of the right lung. Elsewhere many raised pneumonic areas were noted. The branches of the pulmonary arteries showed no gross changes.

Fig. 1.—Low power view of the portal canal showing the narrowed lumen of the hepatic artery (*A*) resulting from proliferation of the intima. Note the increase in periportal connective tissue and cellular elements. The adjacent hepatic lobules are fairly well preserved. Hematoxylin and eosin stain.

There was a recent infarct in the upper pole of the spleen. The cut surface elsewhere revealed prominent malpighian corpuscles. The spleen weighed 195 Gm. The blood vessels showed no gross changes.

The liver weighed 1,450 Gm. Cut sections exhibited an indistinct lobular structure and numerous red areas forming an irregular pattern. The gallbladder, the adrenal glands and the pancreas were grossly normal.

The kidneys were soft and together weighed 440 Gm. The capsule stripped readily, revealing a mottled purple-gray surface with irregular pale yellow areas interspersed among pale gray depressed areas. Scattered throughout the surface were numerous deep purple hemorrhages the size of a pinhead. These hemorrhages extended into the cortex and were somewhat wedge shaped. The cortex and medulla were poorly demarcated; irregular pale yellow areas extended in columns within the pyramids and were mingled with minute hemorrhages. The calices and pelvis renalis were somewhat dilated, and the latter had numerous hemorrhages on the mucosal surface. The ureters were not unusual.

Fig. 2.—High power view of the hepatic artery (fig. 1*A*), showing proliferation of the intima and narrowing of the lumen. The muscle cells within the media are fragmented, and the nuclei are pyknotic. The adventitia is broad and infiltrated with round cells. Hematoxylin and eosin stain.

The gastrointestinal tract and the pelvic organs were normal. The lymph nodes of the mesentery and of the small intestine were moderately enlarged. The mesenteric vessels were grossly normal.

The brain weighed 1,255 Gm. The leptomeninges were edematous, and the blood vessels were injected. The vessels at the base of the brain and the origin of the cranial nerves showed nothing unusual. Grossly the brain appeared edematous, and the cut sections showed no areas of softening or hemorrhage.

Sections through the various levels of the spinal cord revealed no gross abnormalities. Sections of the brachial plexus and femoral nerve appeared normal.

Postmortem cultures from the blood clots within the heart and from the infarcts in the lung and spleen showed Str. haemolyticus.

Microscopic Postmortem Examination.[6]—Heart: The myocardial fibers were spread apart; the nuclei stained well, and the cells appeared normal. The interstitial tissue was edematous, and the coronary vessels showed no changes.

Lung: Sections through the infarcts revealed well demarcated areas replaced by amorphous pink-staining material containing red blood cells, polymorphonuclear leukocytes and large phagocytes harboring dark granules. In the adjacent areas

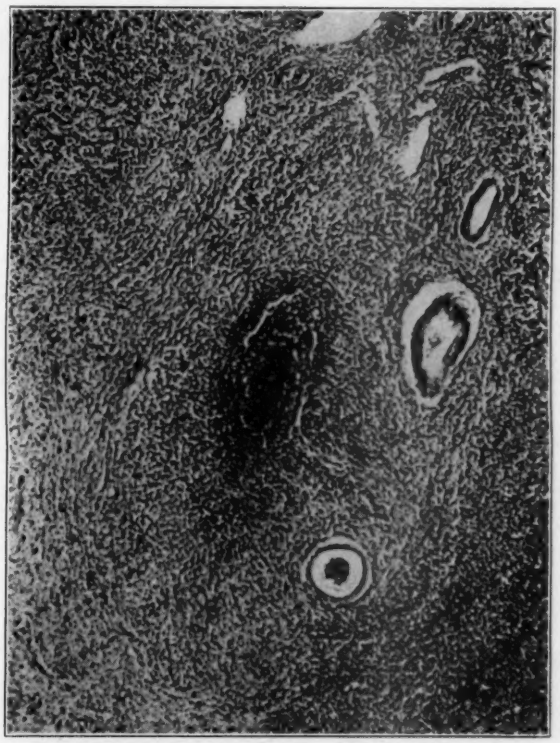

Fig. 3.—Low power view of a recent thrombus within the hepatic artery with destruction of the wall and infiltration with many inflammatory cells. The adjacent hepatic lobules are necrotic. Hematoxylin and eosin stain.

as well as in other sections the alveoli were distended with red blood cells and polymorphonuclear leukocytes; the lining cells were desquamated; the alveolar capillaries were markedly engorged. The bronchioles were filled with similar cells, and the walls were edematous and infiltrated with round cells. The blood vessels showed no changes.

Liver: The capsule was broad and composed of pink-staining bands with few pyknotic nuclei. The central veins were wide, and in places they were surrounded by foci of large phagocytes and polymorphonuclear leukocytes. Within the central portion of the hepatc lobules, the cords were shrunken and the cells were decidedly

vacuolated or fragmented. Here the sinusoids were broad and engorged with blood. Within the peripheral portion of the lobules, the cytoarchitecture was well preserved; the cells stained well, and the nuclei were distinct. The bile capillaries were distended. Foci of round cells and of polymorphonuclear leukocytes were noted throughout the hepatic lobules and extended into the perilobular connective tissue. The portal veins and bile ducts appeared normal. In one section the hepatic artery showed decided proliferation of the intima with narrowing of the lumen (fig. 1). The media stained poorly and contained a few round cells; many muscle fibers were fragmented, and their nuclei were pyknotic; the adventitia was broad and edematous and infiltrated with round cells (fig. 2).

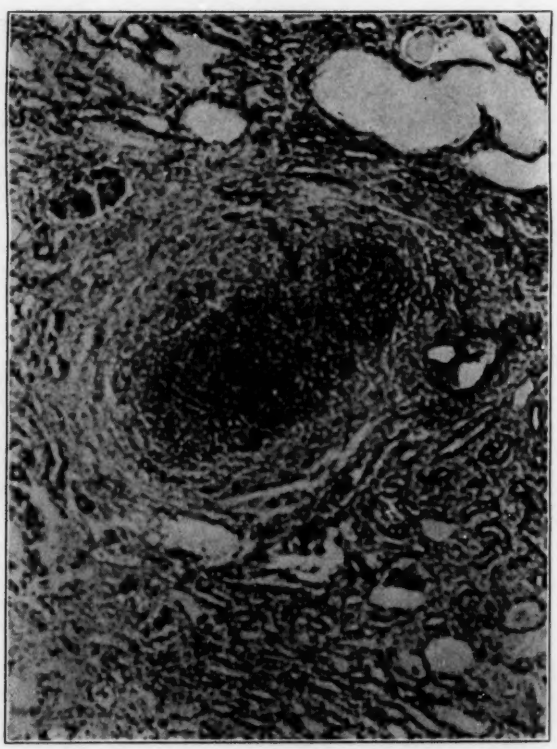

Fig. 4.—Low power view of a recent thrombus within a renal artery. The lumen is occluded, and the wall is destroyed and infiltrated with inflammatory cells. Hematoxylin and eosin stain.

The periportal connective tissue was increased and in places densely infiltrated with round cells and occasional eosinophils. In another section the lobules showed extensive degenerative changes; the cell cords were represented by faintly stained and compressed strands separating engorged and irregular sinusoids. Several hepatic lobules were completely necrotic and were replaced by pink-staining amorphous material and granular debris. Close to these necrotic lobules and within the portal canal the lumen of the hepatic artery was occluded by a recent thrombus; the wall was destroyed in places and densely infiltrated with round

cells and polymorphonuclear leukocytes; the adventitia was edematous and contained numerous inflammatory cells (fig. 3). Fat stain (sudan III) revealed a diffuse distribution of orange-staining material within the liver cells, predominantly within those comprising the central portion of the lobules.

Spleen: The malpighian corpuscles were large, numerous and well demarcated. The pulp sinuses were engorged with red blood cells and numerous phagocytes harboring dark granules. An occasional arteriole revealed an edematous media which stained uniformly pink and few round cells within the wall. In a section through the infarct the pulp was replaced by homogeneous pink-staining material and granular debris.

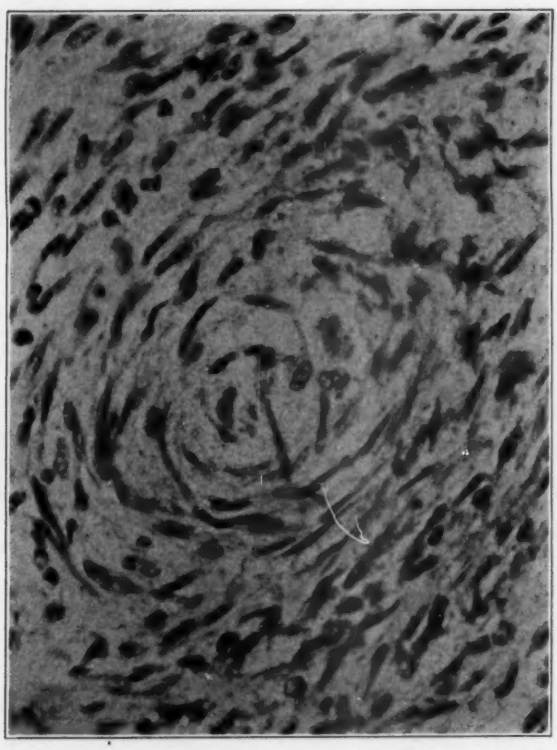

Fig. 5.—High power view of a renal artery showing healing with complete organization. Note the proliferation of young connective tissue cells throughout the wall of the vessel. Hematoxylin and eosin stain.

Kidney: The majority of the glomeruli were well spaced and distinct. The glomerular capillaries were engorged, the subcapsular spaces were clear and Bowman's capsules were intact. Occasional glomeruli were partially or completely replaced by fibrous tissue and surrounded by round cells and polymorphonuclear leukocytes. The tubular structures showed many changes; some of their cells were cuboidal, and the lumens were filled either with red blood cells and polymorphonuclear leukocytes or with amorphous pink-staining material. Groups of tubules were composed of large foamy cells with basally placed nuclei; the lumens were narrow or obliterated. The interstitial tissue was edematous and infiltrated

with round cells, polymorphonuclear leukocytes and occasional eosinophils. The interstitial capillaries were decidedly engorged. The arteries showed various stages of inflammation. In some of the larger arterioles the lumens were completely occluded by recent thrombi with destruction of the intima and media and with infiltration of the entire wall with round cells and polymorphonuclear leukocytes; the adventitia was edematous and showed similar inflammatory cells (fig. 4). In an occasional vessel, beginning organization and canalization were noted (fig. 5). Smaller arterioles showed hyaline necrosis of the media, round cell infiltration and an increase in the adventitia. Around many of these vessels, dense accumulations of round cells extended into the interstitial tissue (fig. 6). Other vessels

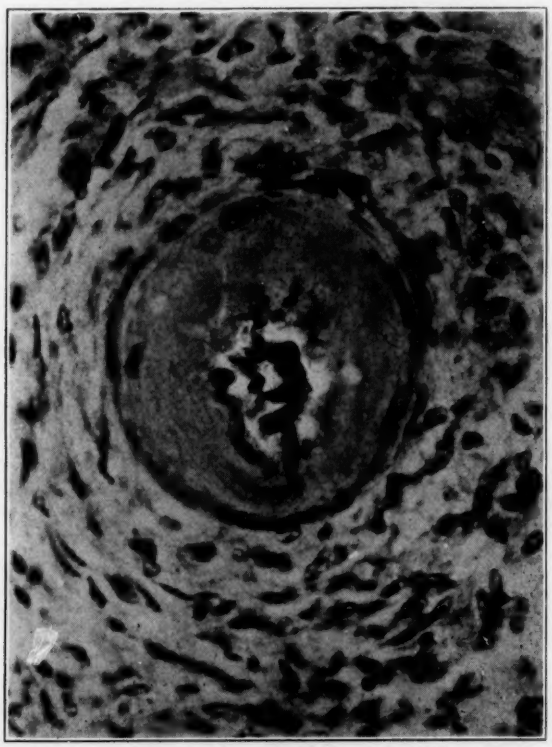

Fig. 6.—High power view of small arterioles within the kidney showing hyaline necrosis of the media, round cell infiltration and edema of the adventitia. Hematoxylin and eosin stain.

showed intimal proliferation with narrowing of the lumen in addition to the changes in the media. Many recent small infarcts were noted within the parenchyma and close to the capsular surface. A stain for fat (sudan III) showed many of the tubular cells laden with orange-staining material.

Brain, Spinal Cord and Nerves: Sections of the brain, spinal cord and nerves were stained with hematoxylin-eosin and sudan III and by Nissl's and Weigert's methods.

The leptomeninges were edematous, and the blood vessels appeared normal. Within the brain the vessels were engorged. There were no perivascular hemor-

rhages or foci of inflammatory cells. The ganglion cells and supporting structures stained well.

Sections of the spinal cord taken at various levels showed no abnormalities. The blood vessels were normal.

Sections from the left brachial plexus revealed profound changes within the nutrient vessels. The lumens of several large arteries were completely occluded by recent thrombi; the intima and media were destroyed, and the entire wall was infiltrated with round cells and polymorphonuclear leukocytes; the adventitia was markedly increased, and many dilated vessels within it gave the appearance of granulation tissue (fig. 7). Many of the smaller arterioles within the connective

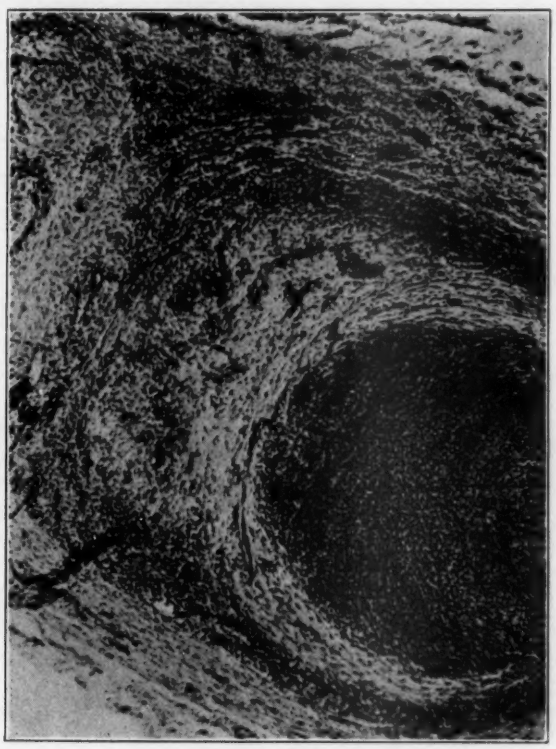

Fig. 7.—Low power view of a large artery within the left brachial plexus showing occlusion by a recent thrombus. The intima and media are destroyed, and the entire wall is infiltrated with inflammatory cells. Note the broad adventitia with decidedly dilated capillaries giving the appearance of granulation tissue. Hematoxylin and eosin stain.

tissue between the nerve bundles showed acute inflammation with infiltration of the wall. Many vessels appeared normal. Study of the nerve bundles revealed moderate edema with occasional foci of round cells. The cells of the sheath of Schwann were increased in number. The blood vessels within the nerve bundles were engorged. Myelin sheath stains revealed that some of the nerve bundles were almost normal, while others showed marked degeneration, as evidenced by

swelling, beading and fragmentation of the myelin. Many of the axis-cylinders were normal; others were fragmented, and some were missing. The epineurium was edematous and in places densely infiltrated with round cells, polymorphonuclear leukocytes and occasional eosinophils (fig. 8).

The right brachial plexus showed similar changes. There appeared to be greater round cell infiltration of the nerve bundles. Again, not all the vessels showed inflammatory changes, nor were all the nerve bundles degenerated. In one of the larger arterioles there was marked proliferation of the intima with narrowing of the lumen in addition to hyaline necrosis of the media and infiltra-

Fig. 8.—High power view of a nerve from the left brachial plexus showing marked degeneration of the nerve fibers with swelling and beading and fragmentation of the myelin. Many of the axis-cylinders are missing. Sudan III stain.

tion of the entire wall. Such changes were not seen at different levels along the same vessel.

The left femoral nerve showed somewhat different vascular involvement. Here the smaller arterioles were involved and revealed hyaline necrosis of the media, sparse cellular infiltration and decided edema of the adventitia with dense perivascular cellular accumulations. Most of the nerve bundles appeared normal at various levels.

The right femoral nerve showed similar changes.

The cervical ganglions appeared normal.

COMMENT

Acute arteritis or periarteritis of small and large arteries associated with necrosis of the media and inflammatory changes within the walls of the vessels have been classified under the term periarteritis nodosa. However, when the relative infrequency of subcutaneous nodules or aneurysms observed clinically or at necropsy is considered, it becomes apparent that the various types of vascular lesions cannot all be placed in such a classification. Whether the lesions are called arteritis, periarteritis, necrotizing or obliterative arteritis or periarteritis nodosa, they all conform to one fairly definite histologic picture. Pathologically, the vascular lesions are characterized by necrosis of the media and fibrinous exudation, by cellular infiltration and the formation of granulation tissue in and about the arterial wall together with varying degrees of proliferation of the intima and by the intimal changes resulting in endothelial desquamation and in a proliferating process which obstructs the lumen. In some vessels, obliteration of the lumen with subsequent canalization results; in others, aneurysmal dilatation of the vessel wall occurs. In either event, important disturbances in circulation result, and infarctions and localized hemorrhage due to aneurysmal rupture are observed. In many cases several stages can be observed in one or more organs which seem to explain clinical remissions and exacerbations. It is characteristic of the disease that not all vessels are included in the inflammatory process and that not all the wall of a vessel is involved.

The literature on periarteritis nodosa has been well summarized in the reports of Schreiber,[7] Lamb,[8] Ophüls,[9] Gruber,[10] Rothstein and Welt,[4] Curtis and Coffey [11] and Middleton and McCarter.[12] Since their reports were made there has been a great increase in the number of

7. Schreiber, R.: Ueber Poliarteritis nodosa, Inaug. Dissert., Königsberg, O. Kümmel, 1904.

8. Lamb, A. R.: Periarteritis Nodosa, Arch. Int. Med. **14**:481 (Oct.) 1914.

9. Ophüls, W.: Periarteritis Acuta Nodosa, Arch. Int. Med. **32**:870 (Dec.) 1923.

10. Gruber, G. B.: Zur pathologischen Anatomie der Periarteritis nodosa, Virchows Arch. f. path. Anat. **245**:123, 1923; Zur Frage der Periarteritis nodosa, mit besonderer Berücksichtigung der Gallenblasen- und Nieren- Beteiligung, ibid. **258**:441, 1925.

11. Curtis, A. C., and Coffey, R. M.: Periarteritis Nodosa, Ann. Int. Med. **7**: 1345, 1934.

12. Middleton, W. S., and McCarter, J. C.: Diagnosis of Periarteritis Nodosa, Am. J. M. Sc. **190**:291, 1935.

cases reported, due probably to the more widespread and thorough knowledge of the character of the disease and to more careful studies at necropsy. The disease occurs predominantly in persons of the male sex between the ages of 3 and 78 years. In Rothstein and Welt's series the incidence was 11.8 per cent in children up to 15 years. The duration of the disease varies up to many months, with periods of remission and exacerbation indicating a tendency to healing. In Spiro's [13] report all arterial lesions found at autopsy had advanced to the stage of healing. In Arkin's [14] interesting case of healed periarteritis nodosa, as demonstrated histologically, the patient lived four years after his one and only acute attack, death resulting from cardiac and renal insufficiency.

Although the disease process may be local (Hauser [15]), the vascular lesions are more often widespread and may occur in certain groups of organs. The resulting clinical pictures have given rise to several syndromes, namely, gastrointestinal, renal, neuromuscular, cardiac, cerebral and neural syndromes. Gruber [16] tabulated the localization of pathologic changes in 108 cases and reported that the kidneys, the liver and the peripheral nerves were involved in 74, 61 and 18.5 per cent of cases respectively, the spleen in 14 per cent and the lungs in 3.7 per cent. The localization within the kidneys is most frequent in this series, and the syndrome of albuminuria, hematuria, edema, retention of nitrogen in the blood and hypertension is a familiar one in the literature.

In the case reported here, following a rather benign course hematuria and albuminuria developed in the third week of illness, associated with retention of nitrogen in the blood and a normal blood pressure. Subsequently there developed marked albuminuria and cylindruria, inversion of the albumin-globulin ratio, fixation of the specific gravity at 1.010 and persistent hypertension. Periods of remission and exacerbation were noted, so that toward the end the amount of nonprotein nitrogen in the blood approached the normal. Pathologically this was not a chronic glomerulonephritis but the result of a fairly

13. Spiro, P.: Zur Kenntnis des Wesens der Periarteritis nodosa, Virchows Arch. f. path. Anat. **227**:1, 1919.

14. Arkin, A.: Clinical and Pathological Study of Periarteritis Nodosa, Am. J. Path. **6**:401, 1930.

15. Hauser, H.: Beitrag zur Frage der Periarteritis nodosa, Frankfurt. Ztschr. f. Path. **36**:22, 1928.

16. Gruber, G. B.: Periarteritis nodosa, Zentralbl. f. Herz- u. Gefässkr. **18**: 145, 1926.

widespread vascular process within the kidneys, varying from medial necrosis with cellular infiltration to thrombosis and even to complete healing with canalization. The changes within the kidneys were a reflection of these varied vascular lesions, and renal examination revealed normal as well as obliterated glomeruli, areas of fibrosis and recent infarctions. The renal syndrome is evident from these pathologic changes, and the presence of acutely inflamed as well as of healed blood vessels accounts for the clinical remissions and exacerbations.

The clinical signs and symptoms of involvement of the peripheral nerves have received much less attention; recently Kernohan and Woltman [17] called attention to the neural manifestations of periarteritis nodosa. Just as changes in the kidneys or other organs are the result of damaged arteries with subsequent thromboses, aneurysms or hemorrhage, likewise changes which occur in the peripheral nervous system are the result of damage or occlusion of the nutrient arteries in the nerve trunks with subsequent degeneration or infarction of the nerve bundles. Lorenz [18] and Meyer [19] described periarteritis of the nutrient vessels of the peripheral nerves. In some of the cases which they studied there was extensive degeneration of the nerves, while in others the nerve tissue was almost normal. Marinesco and Dragenesco [20] reported a case in which the disease involved vessels supplying the peripheral nerves, nerve roots, spinal cord and brain.

Neurologic symptoms are marked by extensive involvement of the peripheral nerves, especially those of the upper and lower extremities. Pain, tenderness of the nerve trunks and muscles, anesthesias, paralyses, loss of reflexes and atrophies occur in irregular distribution. Transient attacks of amblyopia and more permanent disturbances in vision may occur. Ophthalmoscopic examination may reveal thromboses of the central artery, retinal and papillary edema, hemorrhages and detachment of the retina. The meninges and brain may also be involved. The spinal fluid may show increased pressure, xanthochromia, pleocytosis and an increase in total protein content.

17. Kernohan, J. W., and Woltman, H. W.: Periarteritis Nodosa, Arch. Neurol. & Psychiat. **39**:655 (April) 1938.

18. Lorenz, H.: Beitrag zur Kenntnis des multiplen degenerativen Neuritis, Ztschr. f. klin. Med. **18**:493, 1891.

19. Meyer, P. S.: Ueber die klinische Erkenntnis der Periarteritis nodosa und ihre pathologisch-anatomischen Grundlagen, Berl. klin. Wchnschr. **58**:473, 1921.

20. Marinesco, G., and Dragenesco, S.: Sur la forme myelo-neuro-myopathique de la maladie de Kussmaul, Ann. de méd. **22**:154, 1927.

In our case symptoms of polyneuritis occurred on the fortieth day, with vague shooting pains in both upper extremities; later extensive motor and sensory disturbances appeared, associated with areas of anesthesia and with tenderness over the nerve trunks and muscles. The neurologic changes were most marked distally and tended to fade out proximally. The motor disturbances were roughly symmetric, except that the left lower extremity alone was involved. Here the muscle groups, as was the case with the nerves, were more involved distally. Spontaneous improvement occurred during the course of the disease; motor power returned to some extent in both upper extremities, and the left lower extremity became normal. Similarly, wide areas of normal sensation reappeared. The neural syndrome, just as the renal, was the result of changes in the nutrient vessels of the nerve trunks. Various stages of inflammation were observed: cellular infiltration of the wall with slight change in the media, hyaline necrosis of the media with proliferative and desquamative alterations of the intima, thromboses with destruction of the entire wall, "nodosa" formations around the vessel with broad granulation tissue and stages of healing with canalization and proliferation of connective tissue. The latter probably explains the remissions noted clinically. Extensive degenerative changes were noted within the nerve trunks of the brachial plexus with their concomitant sensory and motor disturbances within both upper extremities. Again, some sections showed normal vessels and nerves. This was especially true in the sections from the femoral nerves. The peculiar localizations of the disease process and its tendency to pick out segments of vessels and even portions of the wall at one level make it necessary to examine many sections and sections taken from various portions of the organs.

Because of limitations at necropsy the vessels and nerves of the arms and hands could not be removed for examination and study. The gangrene of the finger tips was most likely the result of inflammatory and thrombotic changes within the vessels supplying the hand.

Clinically, the cranial nerves were normal, there were no meningeal signs and the spinal fluid was normal. Pathologically, no vascular or parenchymal lesions were noted within the brain or the spinal cord.

The occurrence of periarteritis nodosa during the course of scarlet fever, the continuous sepsis after the third week of the disease and the terminal bacteremia due to Str. haemolyticus bring forth some interesting speculations concerning the causes of the disease, in this case at least. What was the role played by the streptococcus? Can one say that both the scarlet fever and the periarteritis nodosa were

manifestations of a bacterial involvement and that the etiologic agent was the same, namely, the streptococcus?

There are a great many theories concerning the causes of periarteritis nodosa. In view of the pathogenesis of the disease process, most writers feel that it is a sepsis. Yet in many of the cases reported, blood cultures and other bacteriologic investigations of material taken from the various organs at necropsy or from subcutaneous nodules failed to show any specific causative agent. In our case a positive blood culture was obtained before death, and postmortem cultures from the blood clots in the heart and from the infarcts of the lung and spleen showed the presence of a similar organism. The temptation is strong, therefore, to accept the "infectious" theory and to attribute the vascular lesions in this case to the same infectious agent as produced the scarlet fever.

The clinical picture may often be so bizarre as to render a correct diagnosis during life extremely difficult. In about 12 per cent of cases subcutaneous nodules are present, and biopsy may be of some value. In the series reported by Rothstein and Welt, diagnosis during life on the basis of definite histologic examination of subcutaneous nodules was made in 8.7 per cent of the cases. Leukocytosis is present in about 32 per cent of the cases, and while eosinophilia is strongly suggestive of periarteritis and the total number of eosinophils may be as high as 79 per cent of the white blood cells, it is present in only 12 per cent of the cases (Middleton and McCarter [12]). On the basis of the frequency of localization, the clinical picture usually includes renal and cardiac insufficiency with their accompanying laboratory data. Death may be due to hemorrhage from the rupture of an aneurysm in a vital area, to necrosis or to insufficiency of a vital organ resulting from vascular involvement.

SUMMARY

Periarteritis nodosa occurred during the course of a mild attack of scarlet fever in an 8 year old boy, who presented an unusual syndrome of nephritis and polyneuritis.

The onset of renal involvement occurred on the twenty-first day of the disease, and signs of peripheral neuritis were evident on the fortieth day.

Although in most cases described a specific etiologic factor or infectious agent has not been proved, the postscarlatinal sepsis, terminal streptococcic bacteremia and postmortem bacteriologic evidence are highly suggestive of a common etiologic agent, namely, the streptococcus.

The term periarteritis nodosa cannot be applied to all the types of vascular lesions, since in only a comparatively small proportion of the cases are subcutaneous nodules observed (12.5 per cent). The disease process, however, is characterized by necrosis of the media and fibrinous exudation, cellular infiltration and formation of granulation tissue in and about the arterial wall together with varying degrees of proliferation of the intima, which in some cases obstructs the lumen. Healing with canalization occurs, and aneurysmal dilatation of the vessel is observed.

The remissions and exacerbations which are observed clinically are explained by the various stages of the vascular lesions and evidences of healing. It is characteristic of the disease that all the vessels are not included in the inflammatory process and that not all the wall of the entire vessel is involved.

Changes which occur in the peripheral nervous system are the result of damage or occlusion of the nutrient arteries in the nerve bundles; hence, changes which occur in the kidneys or other organs are the result of damaged arteries with subsequent thromboses, aneurysms or hemorrhage.

On the basis of the frequency of localization, the clinical picture usually includes renal or cardiac insufficiency. Death may be due to hemorrhage from the rupture of an aneurysm in a vital area, to necrosis or to insufficiency of a vital organ resulting from vascular involvement.

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DISCUSSION

DR. ARCHIBALD HOYNE: A few additional words may be said for the purpose of emphasizing still further the rarity of the coexisting conditions which have been described. For example, since the opening of the Municipal Contagious Disease Hospital there have been about 35,000 cases of scarlet fever, and at the Cook County Contagious Disease Hospital during the same time there have been probably slightly more than half that number; yet no instance of the kind has ever been reported previously from either hospital. Were it not for the interest and industry of Dr. Steiner we should probably have no report at present.

There have been patients with scarlet fever diagnosed as Raynaud's disease, and patients with gangrene described as thromboangiitis obliterans, or as Buerger's disease, but there probably have not been more than 2 or 3 patients in all the years past who resembled this one. One of the important and interesting things in regard to this child was the fact that he had a mild attack of scarlet fever, "a mild attack" that ended in death. This is the sort of thing I think we all encounter frequently: scarlet fever that is looked on as mild, but late in the course of which some complication develops which ultimately results fatally. I think Dr. Steiner has brought out an extremely scientific piece of work.

DR. JOSEPH BRENNEMANN: I think this is an unusally interesting paper well presented. It would take a superclinician to make a diagnosis in a case with such isolated manifestations of periarteritis nodosa unless he had observed a similar case. I feel confident that if Dr. Steiner encountered a case like this again he would probably make the diagnosis. I think it is one of those conditions which one can diagnose more often if one has the condition in mind than if one does not. In a classic case of this sort with well marked nodules that puzzled my associates and me for a considerable time, diagnosis was made after seeing in an article an illustration of cutaneous lesions that resembled closely the lesions in our own case.

DR. MATTHEW M. STEINER: I think I can return Dr. Brennemann's compliment and say that when he encounters another case he will probably make the diagnosis. I think one is fortunate in finding subcutaneous nodules, and even if one does find them, the chances of making the diagnosis are only about 8 per cent.

IMPERFORATE HYMEN CAUSING HYDROCOLPOS, HYDROURETER, HYDRONEPHROSIS AND PYURIA

ITS OCCURRENCE IN AN INFANT

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In the literature I have found only 7 cases of hydrocolpos associated with imperforate hymen and causing symptoms before puberty. In 2 of these cases the children were 6 and 7 years of age, and in 5 the condition occurred at or before 2 months of age. In these reports, which were all gleaned from the European literature, no mention was made of concomitant roentgenographic studies of the urinary tract or of analyses of the blood for evidence of impaired renal function.

In the case reported here, symptoms referable to obstruction of the vaginal outlet appeared within the first few weeks after birth.

REPORT OF A CASE

Diana T. was born on May 11, 1937, of Hungarian parents. The family history revealed nothing abnormal. The child was delivered spontaneously at term and weighed 5 pounds 2 ounces (2,325 Gm.). The obstetrician, Dr. Geza Weitzner, on referring the patient at 8 weeks of age, gave the following antecedent history: The infant's course during the first two weeks, while she was still in the hospital, was uneventful. At home, the parents observed that she was irritable and frequently gave the impression of straining with pain. Since the mother's milk supply was inadequate, the infant was artificially fed, and her unhappy behavior was at first attributed to some minor upset of the alimentary tract. Bouts of diarrhea alternating with constipation further strengthened this interpretation, and several changes in the formula were made. During this time no elevation of temperature was noted. At 6 weeks of age the child had anuria for twelve hours and behaved as though in great pain. Dr. Weitzner found her in severe distress, with tense abdominal walls and bulging, cyanotic perineum but without fever. On catheterization a large quantity of urine was removed. The child acted greatly relieved after this procedure, but a few days later her restlessness and crying spells returned. On my first visit, when the baby was 8 weeks of age, she had frank diarrhea with vomiting but showed no toxic symptoms. One had the impression that she was suffering from severe abdominal pain. Palpation of the abdomen was never satisfactory, since the child always cried and strained during examination. On reduction of the caloric intake the diarrhea ceased within three days. However,

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the infant's disposition did not improve, and fever (temperature, 104.5 F.) developed just at the time when the gastrointestinal symptoms cleared.

The child was admitted to the Babies Hospital at the age of 8½ weeks. The fever seemed to be explained by the finding of a trace of albumin and as many as thirty white blood cells per high power field in the uncentrifuged, catheterized urine, both singly and in clumps, and a pure culture of Aerobacter aerogenes in three successive specimens. Abdominal examination on admission revealed questionable tenderness in the pubic region. The edge of the liver extended 2 cm. below the costal margin in the right nipple line. No mass was identified on external abdominal palpation. The next day several observers felt, by rectal examination, a soft mass on the left

Fig. 1.—Roentgenogram taken after an enema of barium sulfate two days before operation, when the patient was 2 months old.

side between the level of the pubis and the umbilicus, which was interpreted as bulging posterior vaginal wall. It extended as high as the finger could reach. The labia minora looked normal at times and at other times cyanotic. A whitish membrane, which occasionally bulged as though it were the wall of a cyst, was seen between them. Imperforate hymen was diagnosed but was not considered of clinical significance.

Further study of the abdominal tumor and the cause of pyuria was undertaken. A flat plate of the abdomen showed a soft tissue mass which occupied the pelvis and rose several inches above the crest of the ilium. Its upper border, which was in the midline, was sharply demarcated, smooth and convex. An enema of barium sulfate showed the colon to be displaced upward (fig. 1). Intravenous urograms

revealed bilateral hydroureter and bilateral hydronephrosis with obstruction of the ureters at the ureterovesical junction (fig. 2). The bladder was large and situated in the lower right part of the abdomen. The hemoglobin level in the circulating blood was 11.9 Gm.; the red cells numbered 3,200,000 and the white cells 23,500 per cubic millimeter, with 67 per cent polymorphonuclears, 32 per cent lymphocytes and 1 per cent monocytes. The nonprotein nitrogen content of the blood measured 38.2 mg. per hundred cubic centimeters, and the plasma carbon dioxide-combining power was 41.6 volumes per cent. The Mantoux and Kahn tests were negative. A blood culture showing Staphylococcus albus, possibly of the hemolytic type, was considered questionable.

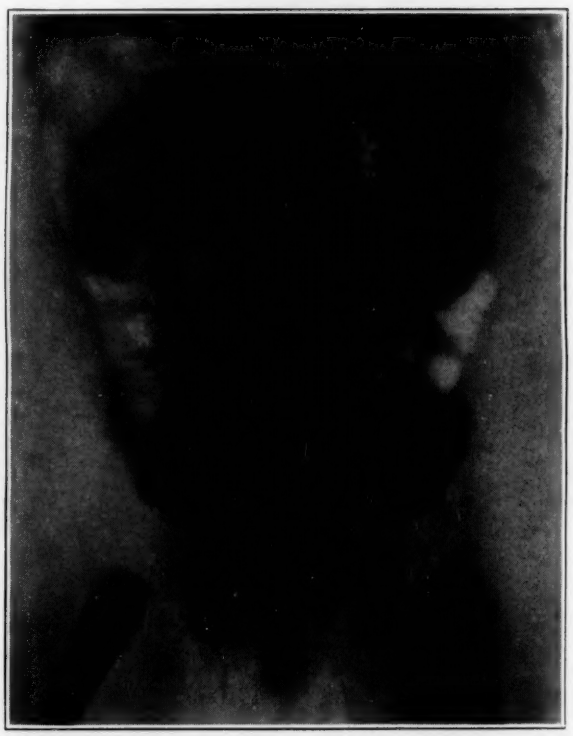

Fig. 2.—Intravenous urogram taken five days before operation, two hours after the injection of the dye.

Because the patient's temperature during the first week in the hospital was above 105 F. daily and because she was rapidly losing ground, laparotomy was performed by Dr. Frederick W. Solly when the patient was 10 weeks of age. The preoperative diagnosis was pelvic tumor of unknown origin causing bilateral hydroureter and hydronephrosis and leading to pyelonephritis from urinary stasis. At operation a cystic tumor was found which almost filled the pelvis, pushing the bladder and the uterus upward and forward. Pressure on this mass caused bulging of the hymen. Before the abdomen was closed, the hymen was incised with release of about 2 ounces (59 cc.) of milky fluid, whereupon the pelvic tumor collapsed. Bacteriologic study of this vaginal fluid yielded no growth. Direct microscopic examination of the material unfortunately was not made.

The infant had a rather stormy postoperative course, with temperature up to 105 F. for about a week and severe diarrhea and meningismus for several days. A. aerogenes persisted in the urine, at times in pure culture and at other times mixed with Staph. albus and Bacillus proteus. The urinary sediment continued to show white blood cells singly and in clumps. Blood culture three days after operation was sterile. Despite nineteen days of treatment with mandelic acid, the child continued to show bacilluria on leaving the hospital, six weeks after operation.

Intravenous pyelography two and five weeks after operation showed some decrease in the dilatation of the ureters and renal pelves. The infant started to gain weight two weeks after operation and did not have any further crying spells or straining after this time. Four months after operation she was readmitted to the hospital for further urogenital study. She was in excellent general condition, but because the catheterized, uncentrifuged urine still showed at times as many as

Fig. 3.—The vagina after 15 cc. of iodized poppyseed oil had been instilled through the introitus vaginae by gravity. The child was held head down during the instillation of the dye and while the picture was taken. The roentgenogram was taken during the time when pyocolpos was diagnosed, that is, when the patient was 22 months old, or twenty months after operation.

ten white blood cells per high power field and on culture yielded B. proteus alone or mixed with Staph. albus haemolyticus and Escherichia communior, a course of treatment with sulfanilamide was given. Again the urine could not be sterilized. The intravenous urogram at this time and also ten months after operation, according to Dr. John Caffey, showed further decrease in the dilatation of the renal pelves and the ureters.

Fifteen months after operation another specimen of catheterized, uncentrifuged urine showed only an occasional leukocyte per high power field and a faint trace of albumin. One culture from the urine yielded Staph. aureus haemolyticus and another was sterile except for one colony of Esch. communior on subculture.

Chemical analysis of the blood showed 22 mg. of nonprotein nitrogen per hundred cubic centimeters, 69 and 82 mg. of sugar, 10.3 mg of calcium, 4.3 mg. of phosphorus and 681 mg. of chlorides. The blood showed 14.2 Gm. of hemoglobin, 5,500,000 red cells and 8,700 white cells, with 25 per cent polymorphonuclear leukocytes. This was the first occasion, fifteen months after operation, that the intravenous urogram showed the upper part of the urinary tract to be normal. In all roentgen films the bladder was small, showing hypertonicity, and there was some irregularity of its borders suggestive of trabeculation in the film exposed fifty minutes after injection of the dye.

At 22 months of age, or twenty months after operation, the patient was readmitted to the Babies Hospital because of a profuse vaginal discharge of ten days' duration after a moderately severe infection of the upper part of the respiratory tract. The parents stated that for the past few months the child's underwear was slightly stained where it came in contact with the vulvar region, but no definite discharge had been seen up to ten days prior to admission. Since this time the discharge increased so rapidly that, on straining, drops of pus were seen to appear in the vulva.

Physical examination on the child's admission to the hospital verified the statement of the parents concerning the amount of vaginal discharge. Although rectal examination did not reveal any mass, during this procedure several cubic centimeters of thick, greenish yellow pus appeared at the introitus vaginae, which was small but definitely patent. This pus yielded Streptococcus haemolyticus beta of the rough variety in pure culture. The catheterized urine was normal microscopically but also yielded streptococci of the same type. The same organism was also found to be the predominating one in the cultures from the nose and throat. The blood count showed 13.6 Gm. of hemoglobin, 4,700,000 red cells and 21,900 white cells, with 79 per cent polymorphonuclear leukocytes, 20 per cent lymphocytes and 1 per cent monocytes. The intravenous urogram again showed no pathologic condition. A roentgenogram taken after 15 cc. of iodized poppyseed oil was instilled by gravity in the vagina, especially in the upper part and in the horizontal direction, showed dilatation (fig. 3). The degree of dilatation was questionable when compared with that in a control case. All this time the child had no fever and her general condition was excellent. The vaginal discharge cleared up in about four days on treatment with sulfanilamide. Ten months has passed since without recurrence of the vaginal abscess.

COMMENT

If the symptoms in this case had been understood preoperatively, a laparotomy could have been avoided. The slowness in arriving at a correct interpretation of the clinical picture may be attributed, at least in some measure, to the infrequency with which imperforate hymen leads to such complications.

As far as the case is understood now, the course of events may be summarized as follows: The infant undoubtedly had a congenitally imperforate hymen. It may be presumed that the occluding membrane was unusually thick to have entirely prevented the escape of the vaginal secretions. One may consider it established that the hymen was completely closed, not only because no discharge was observed but also because the fluid when finally released was sterile. Whether in addition

the rate of secretion was excessive is less certain. Whatever the explanation, it seems that in less than four weeks after the birth of the infant the bulk of the encapsulated vaginal fluid was large enough to cause bouts of diarrhea and constipation, abdominal pain and occasional retention of urine. Probably the coexistent filling of the bladder and intestines determined the degree of the infant's discomfort, since it is hard to conceive of any variation in the size of the hydrocolpos other than progressive enlargement. All of the patient's symptoms, indeed—the crying spells, straining, congestion of the perineum and retention of urine—may be ascribed to the growing hydrocolpos. Eventually this led to bilateral hydronephrosis and hydroureter by interfering with the proper flow of urine. Infection of the urinary tract apparently occurred at the age of 2 months, when the first elevation of temperature was observed.

One often observes that it takes a considerably shorter time for an abnormality to develop than for repair to take place. In the case reported this general principle is well demonstrated. In less than two months hydrocolpos, hydroureter, hydronephrosis and pyuria accompanied by severe constitutional symptoms developed. After removal of the mechanical obstruction it took two weeks for the toxic symptoms to subside and for the abdominal pain, manifested by crying spells, to disappear. However, the pyuria lasted fifteen months and the bacilluria twenty months or longer. Hydroureter and hydronephrosis persisted until fifteen months after laparotomy and hymenotomy. The bladder was described even at this time as showing hypertonicity and trabeculation.

The episode of profuse vaginitis twenty months after operation and the questionably dilated vaginal cavity brings up the problem of possible reaccumulation of pus in the insufficiently draining vagina. Although there is a visible but small hymenal opening at present, it is conceivable that this became insufficient when vaginitis set in, and therefore pyocolpos developed. The large amount of vaginal secretion which was evacuated spontaneously during rectal examination speaks for encapsulated pus and against ordinary vaginitis. Because of the visible vaginal orifice and the rapid response to therapy, no reincision of the hymen was made.

SURVEY OF THE LITERATURE

It is hard to estimate the incidence of uncomplicated imperforate hymen from the literature. Gordon [1] found records of 11 cases observed at the Bellevue Hospital in New York from 1911 to 1931.

1. Gordon, O. A., Jr., in Curtis, A. H.: Obstetrics and Gynecology, Philadelphia, W. B. Saunders Company, 1933, vol. 3, p. 635.

The total number of admissions during this period is not given. In all probability the incidence of imperforate hymen is more frequent than this number would seem to indicate, but because the condition has ordinarily no clinical significance the diagnosis of imperforate hymen is not entered in the record files. Imperforate hymen interfering with the first menstruation by causing hematocolpos appears in the literature as a separate clinical entity. Calvin and Nichamin [2] in 1936 found 28 reported cases. To these they added 14 others from the records of four hospitals. In 1938 Bingham and Blevins [3] added 1 more case to Calvin and Nichamin's series.

Since the patient in the case just reported belongs to a different age group and since the problem here involved hydrocolpos rather than hematocolpos, I have limited my survey of the medical literature to the 7 cases which were similar to mine. Unfortunately the reports are brief and mainly clinical, and therefore it is hard to compare these cases with mine, in which the study of the entire urinary tract revealed considerably greater abnormality than was anticipated.

Melodia's [4] patient was 7 years old and had fever, pain in the pubic region, a palpable tumor in the pelvis, a swollen abdomen, anuria of twenty-four hours' duration and a reddish blue, imperforate hymen. On incision of the hymen 300 cc. of fetid pus which showed varied bacterial flora was released.

The report of Guilleminet and Gayet [5] deals with the case of a 6 year old girl whose initial symptoms were abdominal pain, diarrhea alternating with constipation and retention of urine. The diagnosis of appendicitis having been made, appendectomy was performed, but the symptoms recurred. Finally, on hymenotomy a large amount of pus showing mixed pyogenic flora was released. The symptoms did not recur thereafter.

The patients in the 5 additional cases were 2 months of age or younger. The earliest of these cases is that reported by Godefroy [6] in 1856. The patient was a 2 month old girl who showed a tumor between her labia minora. This mass enlarged on crying, urinating or defecating. On incision of the hymen, which was 2 mm. in thickness and which bled at operation, a teaspoonful of mucus similar to eggwhite was obtained.

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2. Calvin, J. K., and Nichamin, S. J.: Hematocolpos Due to Imperforate Hymen, Am. J. Dis. Child. **51**:832 (April) 1936.

3. Bingham, E. M., and Blevins, W. J., Jr.: California & West. Med. **49**:147 (Aug.) 1938.

4. Melodia, G.: Med. inf., Roma **6**:132, 1935.

5. Guilleminet, and Gayet, R.: Lyon méd. **161**:561 (May 15) 1938.

6. Godefroy, M.: Gaz. d. hôp. **29**:567, 1856.

The next case was reported by Breisky [7] in 1879. He described a full term newborn infant with apparently normal external genitalia except that the posterior part of the hymen protruded as a fluctuating, yellowish white bleb. On incision abundant thick mucus which contained many squamous epithelial cells was found.

In 1900 Bunzel [8] reported a tumor at the vulva of one of his newborn patients. This swelling ruptured spontaneously on the fifth day of life, and some milky fluid was obtained, which, however, was not examined either microscopically or bacteriologically.

The record of the next case was published by Rocher and Balard [9] in 1932. They described an infant 2 days of age who showed enlargement of the vulvar region. The hymen was incised when the patient was 11 days old. A wineglassful of creamy, fetid pus was released, from which colon bacilli and streptococci were grown. Six days later a second hymenotomy was performed, and half a wineglassful of pus was released. After electrocauterization and application of silver nitrate, the hymen remained patent, and pyocolpos did not recur.

The most recent of the 5 cases is that of a patient 25 days of age, reported by Salazar de Sousa [10] in 1934. This infant manifested loss of weight for five days, crying spells, protruding vulva, pyuria and finally anuria for forty-eight hours. Incision of the hymen revealed a huge amount of sterile fluid in the vagina.

Of the 7 cases reported in the literature, in 3 there was no mention of any bacteriologic study of the encapsulated fluid. In Salazar de Sousa's case culture was negative, and the vaginal secretions in the 3 other cases showed varied bacterial flora, colon bacilli, streptococci, staphylococci and pneumobacilli. In only 1 of these 7 cases was there reaccumulation of the vaginal fluid necessitating reincision of the hymen.

SUMMARY

A case is reported in which an imperforate hymen caused hydrocolpos in early infancy, and this in turn, through pressure on the urinary tract, led to bilateral hydroureter and hydronephrosis, pyuria and bacilluria.

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7. Breisky, A.: Die Krankheiten der Vagina, in Billroth, T.: Handbuch der Frauenkrankheiten, Stuttgart, Ferdinand Enke, 1879, vol. 3, p. 595.

8. Bunzel, R.: Prag. med. Wchnschr. **25**:349 (July 26) 1900.

9. Rocher, H. L., and Balard: Bull. Soc. pédiat. de Paris **30**:208 (March) 1932.

10. Salazar de Sousa, C.: Arch. de méd. d. enf. **37**:721 (Dec.) 1934.

THE FEBRILE TYPE OF ERYTHEMA MULTIFORME

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According to Blumer [1] there are two types of erythema multiforme in adults which are of special interest to the internist. The first type, described by Osler [2] in a number of papers, is characterized "by the frequency of recurrences, the association with acute abdominal pain due to exudative processes in the gastro-intestinal tract, frequent inflammatory complications, particularly in the kidney, and the relative high mortality. Occasionally fever may be a prominent feature." The second type, which Blumer particularly emphasized, is characterized by high and long-continued fever and articular pains. The prognosis is usually favorable. Blumer observed 5 adult patients having erythema multiforme of the second type and presenting symptoms of high fever and slight articular pains. He called the condition "the febrile type of erythema multiforme."

The occurrence in children of erythema multiforme of the type described by Blumer has apparently escaped clinical attention, because references to its incidence have not appeared in the literature or in standard textbooks of pediatrics. That it attacks children, though perhaps more infrequently than adults, must be concluded from the fact that in the past twelve years I have encountered 2 cases, each with the characteristic clinical picture of erythema multiforme of the febrile type which has been described as occurring in adults.

These cases are briefly the following:

REPORT OF CASES

CASE 1.—P. N., a 6 year old girl, on May 31, 1935 had fever with pains in the wrists, ankles and legs. The temperature was 102 F., and the throat was congested. There was slight enlargement of the cervical lymph nodes. A rash typical of erythema multiforme appeared on the face, arms and legs. The joints were not tender. The spleen was not enlarged. The remainder of the physical examination gave normal results. The leukocyte count was 22,000, with 72 per cent polymorphonuclears, 19 per cent of which were immature. The erythrocyte count was 4,300,000 and the hemoglobin content (Sahli) was 80 per cent. Urinalysis gave negative results. A blood culture taken at this time showed no growth. During a period of two months the patient had a temperature varying from 100 to 105 F. Mild articular

From the Abraham Jacobi Division for Children, Lenox Hill Hospital.

1. Blumer, G.: Boston M. & S. J. **195**:11, 1926.
2. Osler, W.: Am. J. M. Sc. **110**:629, 1895; **127**:1, 1904.

symptoms involving the wrists and fingers and erythema multiforme appeared at frequent intervals during this period and lasted sometimes but a few hours. Frequent examinations of the blood revealed a slight leukocytosis. There were moderate degrees of anemia and toxemia, which were relieved by blood transfusions. Frequent observations were made during a follow-up period of four years and all showed the patient normal.

CASE 2.—B. L., a 4½ year old girl, on Jan. 1, 1933 suffered an attack of grip. Two weeks later the temperature suddenly rose to 105 F. With this rise in temperature a rash having the characteristics of erythema multiforme appeared over the entire body. The throat was congested. There was some abdominal pain. The rash disappeared after three days, reappearing at short intervals during a month. The leukocyte count was 5,500, with 77 per cent polymorphonuclears, 41 per cent of which were immature. A blood culture showed no growth. The fever lasted for one month and was intermittent, the temperature ranging from 100 F. to 105 F. During this whole time the remainder of the physical examinations and laboratory tests gave entirely negative results. With the subsidence of the fever recovery was complete. The child has remained perfectly well during the past six years.

SUMMARY OF CASES

In these 2 cases the disease was characterized by (1) the appearance of a rash characteristic of erythema multiforme, (2) high intermittent fever lasting for a few weeks to several months or years and (3) very slight pains in the muscles and joints. The causes of the disease are apparently unknown. The causative factor appears to be based on some internal toxic condition. Neither of the 2 children had suffered from tonsillitis, chorea or rheumatism. The disease began with pain in the throat. The evanescent appearance of the erythema was characteristic. It appeared on various parts of the body and usually disappeared after a few days, only to reappear in another area. The points of predilection were the extensor surfaces of the extremities. The course of the disease was very stormy, sometimes lasting for many months. The patients became entirely free from symptoms, one after one month, and the other after two months, of high fever. The patients, both of whom were girls, have been under observation from four to six years. Laboratory tests including blood cultures were negative. In the differential diagnosis such conditions as grip, pneumonia, rheumatic fever and sepsis were considered by various colleagues who saw the patients in consultation.

At times Still's disease, when an erythematous rash is present with high intermittent fever and articular pains, may simulate the febrile type of erythema multiforme. Sometimes only after many months' observation the diagnosis will be made clear by the presence of swelling of the joints and by pathologic conditions revealed by roentgenograms. The following case of Still's disease at first caused this difficulty in diagnosis:

B. S., a 10 year old girl, on Nov. 25, 1927 suddenly complained of pain in the toes, wrists and arms. The temperature was 102 F. A rash typical of

erythema multiforme was present on the arms, face and legs. The throat was congested. The cervical glands were palpable. The temperature varied between 100 F. and 104.6 F. and continued for two months. The erythema disappeared after three days but appeared again after short intervals. The leukocyte count was 9,200, with 63 per cent polymorphonuclears. The erythrocyte count was 4,200,000, and the hemoglobin content was 70 per cent. The blood culture was negative. During the next few months there were a few periods of normal temperature, but during most of the time the rash appeared at intervals, with a rise of temperature as high as 104 F. and slight articular pains. These symptoms continued intermittently during the following year. At this time roentgenographic examination of the hands and wrists showed evidence of involvement of the periarticular structures. The physical examination and other laboratory tests gave entirely negative results. During the next few years the articular pains and the rash appeared from time to time, with a slight rise in temperature. These symptoms have gradually become less severe in intensity after nine years' duration. The examination of the heart has always given normal results. Observation eleven years after the onset of this condition revealed normal physical conditions except for slight spindle-shaped deformities of the fingers, wrists and toes. Roentgenograms made at this time revealed the following conditions: There was evidence of an old infectious arthritic process involving both wrists which had developed into ankylosis; all of the intracarpal articulations had been obliterated and there was evidence of some fusion bilaterally, between the metacarpal bones and the distal row of carpal bones; the articular spaces between the radius and the carpal bones on both sides were considerably narrowed; atrophic arthritic changes were also present between several of the interphalangeal articulations, associated with fusiform swelling of the periarticular soft tissues. The roentgenographic appearance was that of a chronic infectious arthritis, probably Still's disease.

Greenwald [3] has recently reported a case of a boy 8 years of age with high intermittent fever lasting over sixteen months, articular pains and a rash. Physical examination gave negative results except for revealing "generalized hypersensitiveness" and an erythematous rash. The condition was finally diagnosed as Still's disease.

Dr. Emanuel Libman, who saw the first patient in consultation, called my attention to the syndrome which I have described. Having also examined the other patient, he concurred in the opinion that her disease was of the same kind, erythema multiforme of the febrile type.

CONCLUSION

Two children who had erythema multiforme with high intermittent fever and mild articular symptoms made a complete recovery without complications. The disease in these 2 patients resembled closely that described by Blumer as occurring in adults and called by him "the febrile type of erythema multiforme."

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3. Greenwald, H. M.: A Case of Prolonged Hyperpyrexia, Am. J. Dis. Child. **53**:250 (Jan.) 1937.

Progress in Pediatrics

CIRRHOSIS OF THE LIVER IN CHILDREN

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Cirrhosis of the liver, one of the rarer diseases of childhood, occurs more frequently than is generally recognized. Emphasis in the past has been placed on cases in which autopsy was done; few patients proved to have cirrhosis have been followed clinically for years. The clinical changes observed depend on the extent of the damage to the liver and the consequent reduction of hepatic efficiency. The disease in children usually differs from that in adults only in having a more acute onset and more rapid course. We have studied 20 children with cirrhosis, 13 of whom came to autopsy. Two patients, 5 years and 4 months of age respectively, who were proved by biopsy of the liver to have cirrhosis, have survived three and five years thus far. Studies of hepatic function have been done recently on them (table 1).

The identification of the etiologic agent in cirrhosis in human beings usually is impossible. Many attempts have been made to reproduce the lesions [1] in experimental animals. The etiologic factors so far recognized may be outlined thus:

Intoxication of cells by:
- Metals
- Organic compounds

Obstruction to bile by:
- Congenital defects
- Tumors, stones

Obstruction to blood by:
- Cardiac failure
- Venous thrombosis

Infection:
- Of blood stream
- Of biliary tract
- Through effects of toxins

From the Departments of Pathology and Pediatrics, Duke University School of Medicine and Duke Hospital.

1. Moon, V. H.: Experimental Cirrhosis in Relation to Human Cirrhosis, Arch. Path. **18**:381 (Sept.) 1934.

Parasitism of:
- Blood
- Intestinal tract

Diet and metabolic disturbance by:
- Alcohol
- Diabetes, hyperthyroidism

Combinations of:
- Intoxication and infection
- Obstruction and infection

Unknown causes

The following cases illustrate many of these factors, but the demonstration of etiologic agents often was impossible. The cases are grouped by the clinical facts or by the resemblance to lesions ascribed to known specific factors.

CASE 1.—A 3½ year old white girl,[2] admitted to the McLeod Infirmary, Florence, S. C.,[3] had been well except for mild infections of the respiratory tract and gastric upsets. Three days before admission she had malaise and icterus was noted; no history of infection or medication was obtained. She was well developed and somnolent, with dilated superficial abdominal veins. The temperature rose; the jaundice became deeper; ascites, hematemesis and melena developed. She died on the tenth day.

At autopsy (Dr. Julian P. Price), the abdomen contained 750 cc. of dark yellow fluid. The liver was small, yellow and soft and contained nodules measuring 0.5 to 2.5 cm. The splenic capsule was scarred. A thrombus in the portal vein was traced into the liver. The liver showed tremendous necrosis in the central and middle zones, with collapse of the tissue. A few lymphocytes and polymorphonuclear leukocytes were present, and new bile ducts were forming.

This patient had acute necrosis, with scarring and beginning regeneration resembling that caused by heavy metals; the agent was unidentified. The thrombosis in the portal vein probably followed the necrosis or secondary infection and was consistent with Banti's syndrome.[4]

CASE 2.—One month before admission an enlarged abdomen, anorexia, vomiting and diarrhea developed in a 17 month old boy. He was poorly nourished, acutely ill and sallow but not jaundiced. The cervical, axillary and inguinal nodes were enlarged, the tonsils injected and the abdomen tympanitic. The smooth, sharp edge of the liver extended 5 cm. below the costal margin; the hard, smooth spleen extended to the umbilicus. Marked jaundice, edema, ascitic and pleural fluid developed. He died on the sixth day.

At autopsy the abdomen contained 500 cc. of clear fluid. The liver was enlarged and yellow, with nodules measuring 3 to 4 mm. (fig. 1 *A*). Micrococcus catarrhalis was cultured from the lung, spleen and pleural fluids. The hepatic lobules were disorganized in the central and middle zones (fig. 2) by degeneration

2. Price, J. P.: Acute Necrosis of the Liver, Am. J. Dis. Child. **48**:586 (Sept.) 1934.

3. All patients were admitted to Duke Hospital unless otherwise noted.

4. Winternitz, M. C.: The Effect of Occlusion of the Various Hepatic Vessels upon the Liver, Bull. Johns Hopkins Hosp. **22**:396, 1911.

of cells which showed coarse hyaline granules (fig. 3). In the increased young and old connective tissue, lymphocytes and a few macrophages and polymorphonuclear leukocytes were scattered. All secondary lymphoid follicles in the body showed hyalinization. Interstitial mononuclear pneumonia with hemorrhage was present. The renal tubules contained fat.

This patient had acute portal cirrhosis resembling that produced by a toxic substance.[5] Evidence of a terminal infection was present in the lungs and kidneys.

The metals or elements producing the most pronounced lesions experimentally are phosphorus, copper, lead, manganese, silica, thorium,

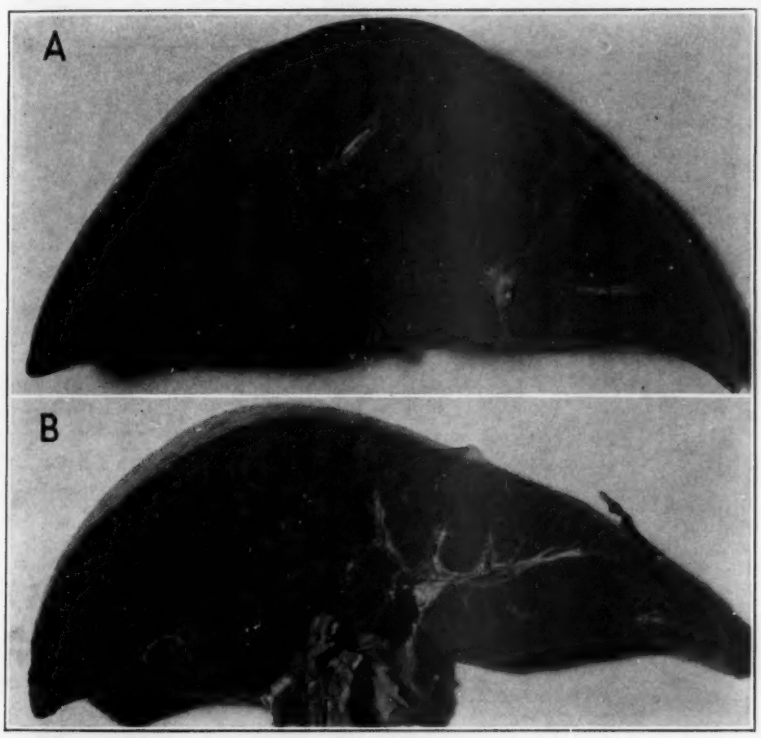

Fig. 1.—*A* (case 2), fine cirrhosis in a 17 month old infant, resembling that caused by heavy metals. *B* (case 3), coarse cirrhosis of unknown cause in a 12 year old boy, with necrosis of the regenerated nodules following operation under ether anesthesia.

cobalt, nickel and arsenic. Only the last is encountered commonly in man.[1] Acute poisoning results in massive destruction of hepatic cells and rapid progression to death from acute yellow atrophy; chronic

5. Dr. F. B. Mallory examined sections and thought the lesion consistent with cirrhosis due to lead. No history of exposure to heavy metals was obtained. Dr. Haywood M. Taylor, of the department of biochemistry, found 0.01075 mg. of lead per gram of liver and approximately equal amounts in 5 controls.

Case, Initials, Date	Age,* Race, Sex	Duration of Illness	Autopsy	Infections: Age*	Infections: Variety	Poisons	Temperature Peak, C.	Hemorrhage: G. I.	Hemorrhage: Skin	Ascites	Jaundice	Collateral Circulation	Hepatomegaly
1 Z. C. 10/1/31	3½ W F	3 days	+	1 to 3	Respiratory	0	38.8	+	0	+++	++++	++	0
2 D. W. 10/21/37	17 mo. W M	1 mo.	+	....	0	0	39.5	0	0	++	+++	0	+++
3 M. G. 1/23/35	12 W M	6 mo.	+	11 11	Mumps Pertussis	0	39	0	0	++++	+++	0	+++
4 M. J. 1/25/38	4 mo. W M	4 mo.	0	..	0	0	39	+	0	++	++++	+++	++++
5 W. S. 12/10/30	16 N M	3 yr.	+	13	Rheumatic fever	0	38.8	0	0	±	0	0	+
6 E. D. 2/17/32	8 N M	?	+	8 8	Intestinal Otitis	0	38.1	+	0	0	0	0	0
7 N. W. 12/19/32	15 N M	4 yr.	0	 15	Measles Pertussis Abscessed teeth	0	....	0	0	+++	0	..	+++
8 A. W. 7/23/33	15 W M	5 yr.	+	1 6 7 8 5 to 15	Intestinal Measles Varicella Pertussis Tonsillitis	0	38.5	++++	+++	+	0	0	++++
9 C. B. 6/20/36	11 W M	2 yr.	..	2, 3, 5 4 5 6 9	Otitis, pneumonia Measles Mumps Pertussis Tonsillitis	0	38	+++	0	P. H.	0	0	0
4/14/37	12	3 yr.	‖	11	Otitis	0	37	0	0	0	0	0	0
10 L. S. 4/30/38	16 W F	5 mo.	+	 16	Measles Otitis Respiratory	0	39.7	0	0	+++	++++	0	0
11 H. J. 6/23/31	7 wk. N M	1 wk.	+	6 wk.	Respiratory	Castor oil	36.8	+	+++	0	+++	0	+++
12 W. B. 1/10/37	6 wk. N F	6 wk.	+	Birth	Respiratory	0	37.8	0	0	++	0	0	++++
13 B. W. 12/4/37	6 hr. N F	.....	+	..	0	0	36	0	0	0	0	0	+++
14 E. H. 11/7/36	5 W M	2 mo.	..	2 3 5	Measles Pertussis Tonsillitis	0	39.5	+	0	++++	++	++	0
11/29/38	7	2 yr.	..	..	.....	0	36.6	0	0	0	0	..	0
8/24/39	8	3 yr.	‖	8	Pyelitis	0	37.2	0	0	0	0	++	0
15 A. B. 4/9/36	9 W M	.....	0	8 9	Malaria Hookworm	0	39.5	++++	+	++++	0	0	+++
16 L. L. 4/17/34	4 mo. W F	4 mo.	+	2 mo.	Pneumonia	0	42	0	0	0	++++	0	+++
17 W. B. 2/11/39	6 N M	1 mo.	+	3 5 5	Urinary Measles Pertussis	Anthelmintics	40.4	0	0	++++	++++	0	+++
18 M. S. 1/17/34	6 W F	4½ yr.	+	1½ 6 6	Influenza Tonsillitis Ascaris	0	38	+	0	++++	++++	0	++++
19 R. D. 3/26/34	4 mo. W F	1 wk.	..	3 mo.	Respiratory	0	38	0	0	0	0	0	++++
3/13/35	15 mo.	1 yr.	..	1 yr.	Urinary	0	40	0	0	0	0	+	+++
11/28/38	4½	4½ yr.	..	..	.....	0	38.5	0	+++	0	0	++	++
7/27/39	5½	5½ yr.	..	..	.....	0	36.5	0	0	0	0	++	++
9/ 5/39	5½	5½ yr.	‖	..	.....	0	....	0	0	0	0	++	+
20 C. A. 5/18/34	12 W M	4 mo.	..	2 3 11	Pertussis Mumps Varicella	0	37.8	++++	0	0	0	+	+
		1 yr.	0	13	.....	.....	....	?	..	..	..	..	..

* Years unless noted.
† D = direct, I = indirect reaction.
‡ Icteric index 180.
§ Culture at autopsy.
‖ Still alive.

Hemoglobin, Gm. per 100 Cc.	Red Blood Count, Millions	White Blood Count	Fragility of Red Blood Cells: Hemolysis, % NaCl in Solution: Start	Fragility of Red Blood Cells: Hemolysis, % NaCl in Solution: Complete	Van den Bergh Reaction †	Bilirubin, Mg. per 100 Cc.	Four Hour Bilirubin Retention, Percentage	Cholesterol, Mg. per 100 Cc.	Esters, Percentage	Bromsulphalein Retention, Percentage: 5 Min.	Bromsulphalein Retention, Percentage: 30 Min.	Galactose Excretion, Gm. in 6 Hr.	Total Proteins, Gm. per 100 Cc.	Albumin, Gm. per 100 Cc.	Globulin, Gm. per 100 Cc.	Bacteria and Site of Culture
6.7	....	15,000	....	....	..	‡	....	...	..	..	..	...	....	....	....	
4.3	3.10	10,050	0.42	0.28	D	24.6	....	150	..	..	..	...	5.3	1.9	3.4	Micrococcus catarrhalis; lung, pleurae, spleen §
10.4	2.80	8,040	0.38	0.32	D	6.0	....	...	..	..	..	6.0	6.69	1.59	5.10	Sterile; abdomen
12.1	3.96	8,040	0.42	0.32	D	14.4	....	...	..	..	..	...	6.0	1.8	4.2	Staphylococcus aureus; throat
14.0	5.12	29,600	....	....	..	....	....	...	..	..	..	...	....	....	....	Pneumococcus; blood
7.5	1.20	17,600	....	....	..	....	....	...	..	..	..	...	....	....	..,.	Sterile; blood Sterile; heart, intestine §
11.0	3.91	8,900	....	....	..	0	....	...	..	..	0	...	....	....	....	
6.5	2.40	7,500	....	....	I	0.2	....	147	..	..	..	...	6.47	2.68	3.79	Beta streptococcus; throat Sterile; blood
4.6	1.80	8,100	....	....	I	0.1	....	...	..	15	0	...	4.97	....	....	
12.4	3.70	9,800	....	....	..	....	....	...	..	..	..	...	....	....	....	
12.4	3.50	9,200	....	....	..	....	....	...	..	..	..	...	6.9	2.6	4.3	Escherichia coli; peritoneum
+	....	.....	....	....	..	....	....	...	..	..	..	...	....	....	....	
+ 7.0	1.98	11,560	....	....	..	....	....	...	..	..	..	...	....	....	....	
+	....	.....	....	....	..	....	....	...	..	..	..	...	....	....	....	
10.9	3.32	15,160	0.44	0.38	D	1.7	....	...	..	35	15	0.5	4.9	2.0	2.9	Sterile; gallbladder
11.0	4.39	7,600	....	....	..	0	....	145	76	..	..	...	6.4	3.7	2.7	
11.8	3.87	10,100	0.40	0.32	..	0.2	21.4	...	..	8	0	0	6.9	4.4	2.5	
10.8	3.25	11,000	....	....	..	....	....	...	..	..	..	...	....	....	....	
12.7	4.00	21,300	....	....	D	23.0	....	...	..	..	..	...	....	....	....	No streptococci or Klebs-Loeffler bacilli; throat Escherichia coli; lung § Sterile; liver §
+ 12.4	3.89	4,720	>0.50	<0.30	D	41.6	....	146	20.5	..	..	...	6.5	2.6	3.9	
6.2	2.07	10,200	....	....	..	....	....	...	..	..	..	...	....	....	....	
11.6	3.60	4,700	....	....	..	....	....	...	..	..	..	...	....	....	....	
10.8	4.00	9,240	....	....	..	0	....	231	..	30	..	...	6.46	3.21	3.25	Staphylococcus aureus; incision
10.4	3.09	20,000	....	....	1	Trace	....	155	76	3	0	...	7.3	4.4	2.9	Staphylococcus albus; nose
12.7	....	.....	....	....	..	....	0	...	..	..	..	...	6.9	4.4	2.5	
11.8	4.08	15,300	0.44	0.30	..	....	....	...	..	..	..	0	....	....	....	
8.0	3.45	3,280	0.42	0.34	I	Trace	....	...	..	35	0	...	....	....	....	

poisoning results in gradual scattered destruction of cells with repair and delayed death. The picture in children is not complicated by age and the acquired resistance of surviving or regenerated cells to repeated exposures to the same poison.[6]

CASE 3.—Six months before admission pain developed in the epigastrium of a 12 year old boy, with nausea, vomiting and occasional diarrhea. Three weeks

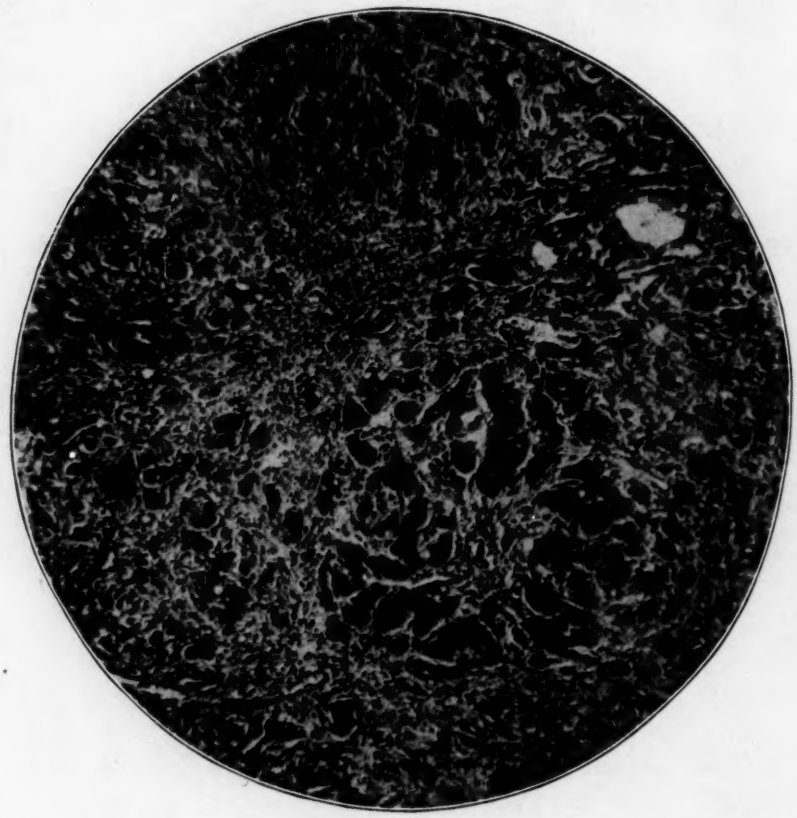

Fig. 2 (case 2).—Active cirrhosis in a 17 month old infant, resembling that caused by heavy metals. A periportal area is seen at the upper right. Hematoxylin and eosin stain; × 90.

before admission ascites and jaundice were noted. He was undernourished, with pharyngitis, an apical cardiac friction rub and a tender abdomen. The liver extended 4 fingerbreadths below the costal margin; the tip of the spleen was felt. Twenty-six hundred cubic centimeters of ascitic and 90 cc. of pleural fluid (from the left side) were removed. Twelve hours after biopsy of the liver he died.

6. MacNider, W. deB.: A Consideration of the Acquired Resistance of Fixed Tissue Cells to Injury, Ann. Int. Med. **12**:147, 1938.

At autopsy, the liver was composed of jaundiced nodules measuring 5 mm. separated by septums of fibrous tissue from 3 to 4 mm. thick (fig. 1 *B*). Enlarged lymph nodes were found at the hilus of the liver and in the mesentery. Both lungs showed signs of fresh pleurisy.

The liver was composed of isolated regenerated nodules separated by dense old fibrous tissue, in which new bile ducts were proliferating at the periportal areas. Necrosis had greatly progressed during the time between biopsy and autopsy.

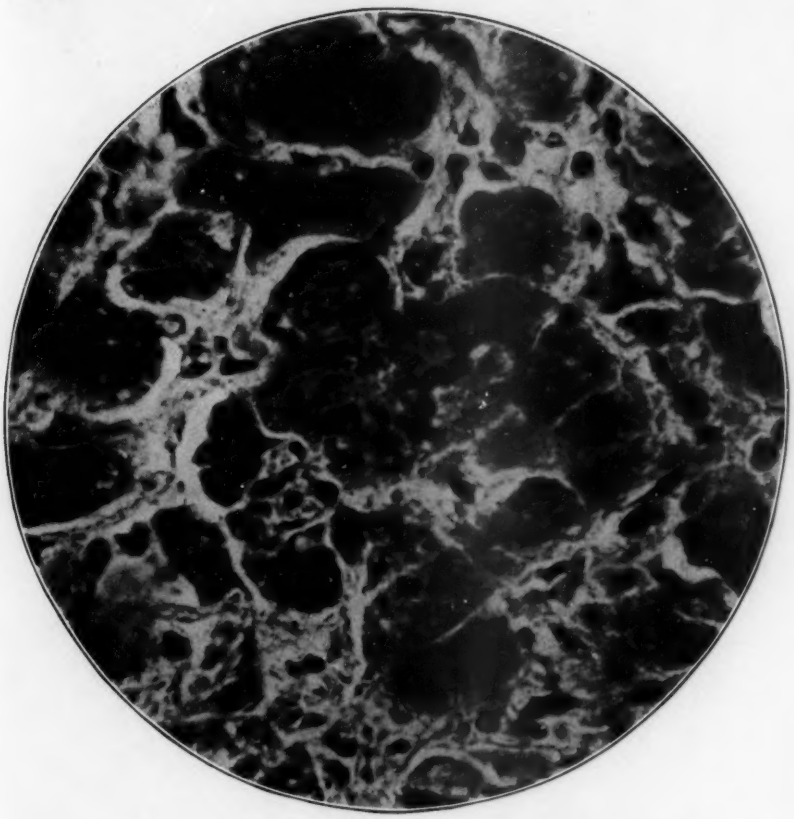

Fig. 3 (case 2).—Hepatic cells showing coarse particles of refractile "alcoholic hyalin" at the center of the field. Hematoxylin and eosin stain; × 1125.

This patient had long-standing portal cirrhosis of unknown cause and duration, with recent exacerbation and subacute necrosis of the regenerated nodules. The terminal necrosis may have been hastened by the anesthesia (ether).

The organic poisons mentioned by various observers include ethyl alcohol, chloroform, carbon tetrachloride and related substances, tar and cinchophen. Anaphylactic shock in animals from injections of foreign protein can result in cirrhosis (see case 9 for exacerbation due to typhoid

vaccine). The end result of damage by organic and inorganic substances is the so-called portal (Laennec's, atrophic) cirrhosis.[1]

CASE 4.—A 4 month old boy had been born three weeks prematurely, with jaundice and an enlarged spleen and liver. Nosebleeds, hematuria and bloody and light-colored stools developed. The hard, nodular liver extended 6 cm. and the firm spleen 7 cm. below the costal margin. Dilated abdominal veins and ascites were found. A specimen for biopsy was taken of the extremely cirrhotic liver.

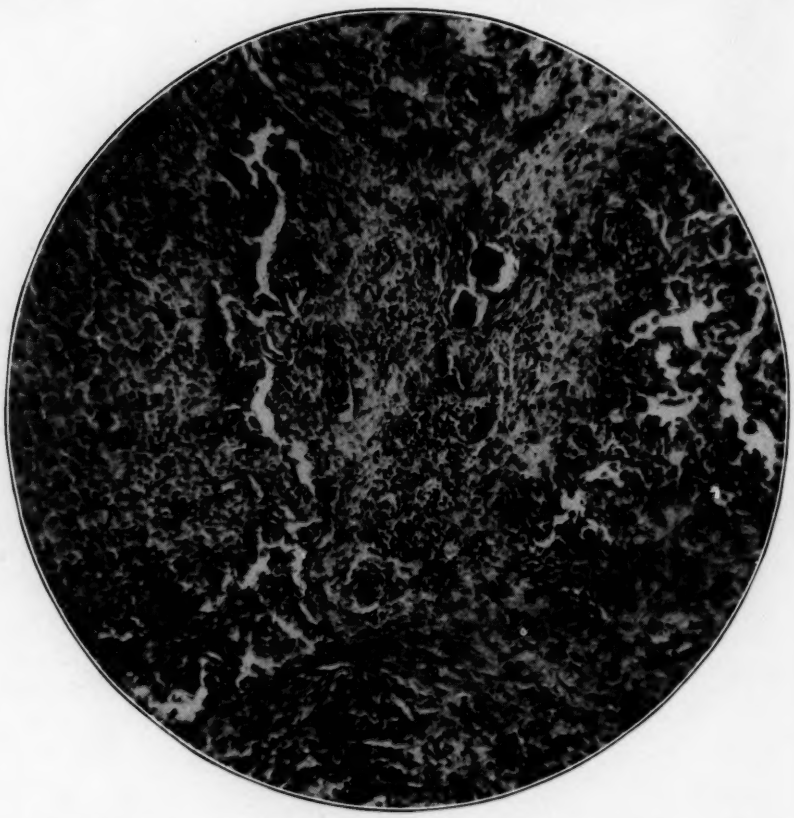

Fig. 4 (case 4).—Cirrhosis in a 4 month old infant as a result of congenital biliary obstruction. A nodule of hepatic cells is seen at the left, a periportal area is in the center, with inspissated bile in small bile ducts; a larger bile duct is at the right, lined with hypertrophied flattened epithelium. Hematoxylin and eosin stain; × 90.

The gallbladder was atrophied and fibrous and contained a small cyst; the common duct was normal in size. Icteric ascites, which require tapping, and pleural fluid persisted. He died two months later, and autopsy was not performed.

The isolated hepatic nodules were separated by large areas of old fibrous tissue containing small dilated bile ducts lined with flattened atypical epithelium and filled with inspissated bile (fig. 4).

This patient had congenital obstruction at an undemonstrated site in the biliary tract, with no superimposed infection.[7]

Malformation of the bile ducts results in changes in the liver comparable to those in animals after ligation of the common duct. In adults, obstruction to bile by tumors of the pancreas or ampulla of Vater, stone in the common duct or benign stricture[8] results in so-called biliary cirrhosis.

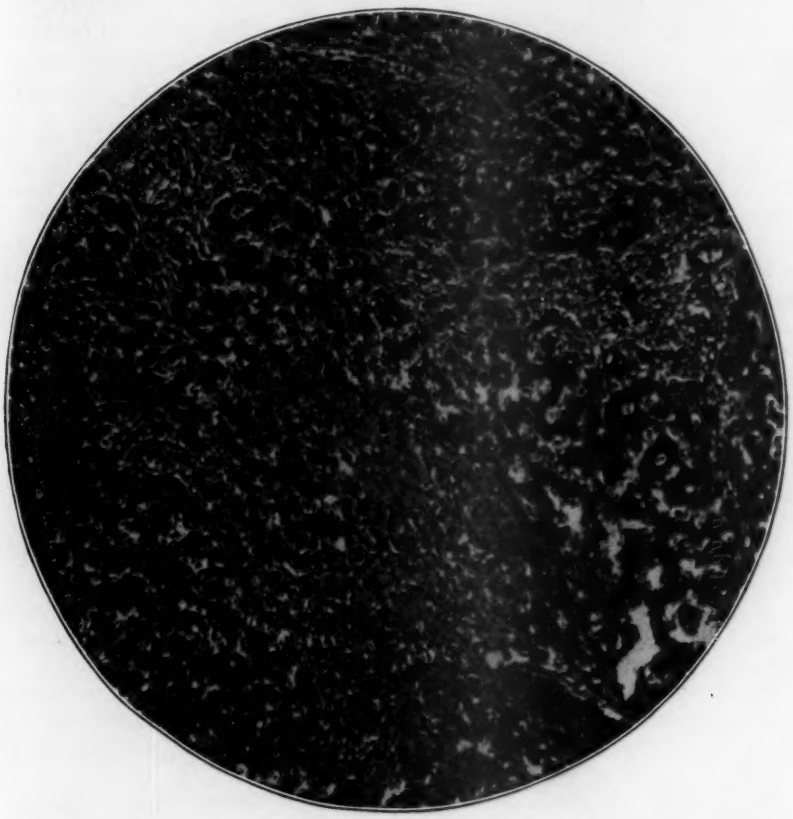

Fig. 5 (case 5).—Central scarring in a 16 year old boy following prolonged cardiac failure. A medium-sized vein is seen at the lower right, with periportal areas at the upper right and left and center left. Hematoxylin and eosin stain; × 90.

CASE 5.—Three years before admission a 16 year old Negro had had fever, with pain and swelling of the joints. Each winter he was bedridden with edema

7. Since this paper was prepared, a 4 month old white girl with identical findings at biopsy has been studied.

8. MacMahon, H. E., and Mallory, F. B.: Obstructive Cirrhosis, Am. J. Path. **5**:645, 1929.

and cough, which had recurred one month before admission. He was orthopneic, with signs of mitral stenosis, abdominal distention but little peripheral edema. Pericarditis developed and he died on the sixth day.

At autopsy, the liver was enlarged and fibrous, with chronic passive congestion. The central areas showed atrophy of the cells and increase in fibrous tissue (fig. 5).

This patient had chronic rheumatic heart disease, with long-standing cardiac failure and central scarring of the liver.

Prolonged stasis of venous blood from cardiac failure,[9] thrombophlebitis of the inferior vena cava[10] or infection with Schistosoma mansoni[11] can produce fibrosis which is most marked in the central areas—cardiac or circulatory cirrhosis.

CASE 6.—Two weeks before admission to Lincoln Hospital, Durham, cramping umbilical pain, with bloody diarrhea developed in an 8 year old Negro. He was acutely ill, emaciated and dehydrated, with purulent otitis media. The Widal reaction was negative. He died after twenty-four hours.

At autopsy, the liver showed diffuse scarring, with yellow nodules measuring from 1 to 2 mm. (fig. 6*A*). In the cecum and ascending colon were numerous ragged hemorrhagic ulcers, from which no typhoid or dysentery bacilli were grown. The liver was composed of regenerated nodules, with scattered early necrosis. Old fibrous tissue infiltrated with lymphocytes was marked around the bile ducts and minimal between the nodules; rare new bile ducts were present. The lesions in the intestines and mesenteric nodes resembled those of typhoid.

The old lesions in the liver probably represented a chronic infectious hepatitis which had metastasized from the intestine through the portal circulation. The necrosis resulted from the recent exacerbation at the onset of clinical typhoid fever.

CASE 7.—For four years a 15 year old Negro, admitted to L. Richardson Memorial Hospital, Greensboro, N. C., had had intermittent swelling of the abdomen, indigestion and increasing weakness; ascites developed. Two abscessed teeth were removed, and a specimen for biopsy taken of the hard, granular liver, which was twice normal size. Jaundice developed and he died after one month. Autopsy was not performed.

The hepatic lobules were distorted by bands of old fibrous tissue which ran irregularly through the central and middle zones, and they contained scattered lymphocytes and a few new bile ducts. The cells were separated by fine fibrous strands.

This patient had slowly progressive scarring of unknown cause, with little preoperative hepatic insufficiency. The diffuse character suggested an infectious origin; three Wassermann tests were negative.

CASE 8.—A 15 year old boy had had sore throat once each winter. Three weeks before admission he had epistaxis, and two weeks later the gums began to

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9. Boland, E. W., and Willius, F. A.: Changes in the Liver Produced by Chronic Passive Congestion, Arch. Int. Med. **62**:723 (Nov.) 1938.

10. Rigdon, R. H.: On the Relation Between Thrombophlebitis of the Inferior Vena Cava and Occlusion of the Hepatic Veins (Endophlebitis Hepatica Obliterans), Bull. Johns Hopkins Hosp. **53**:162, 1933.

11. Rousselot, L. M.: The Role of Congestion (Portal Hypertension) in So-Called Banti's Syndrome, J. A. M. A. **107**:1788 (Nov. 28) 1936.

bleed. He was acutely ill, with petechiae on the skin, enlargement of the superficial lymph nodes and icteric scleras. The smooth, soft liver extended 8 cm., and the firm, nontender spleen 7 cm., below the costal margin. He died on the ninth day from anuria and rectal hemorrhage.

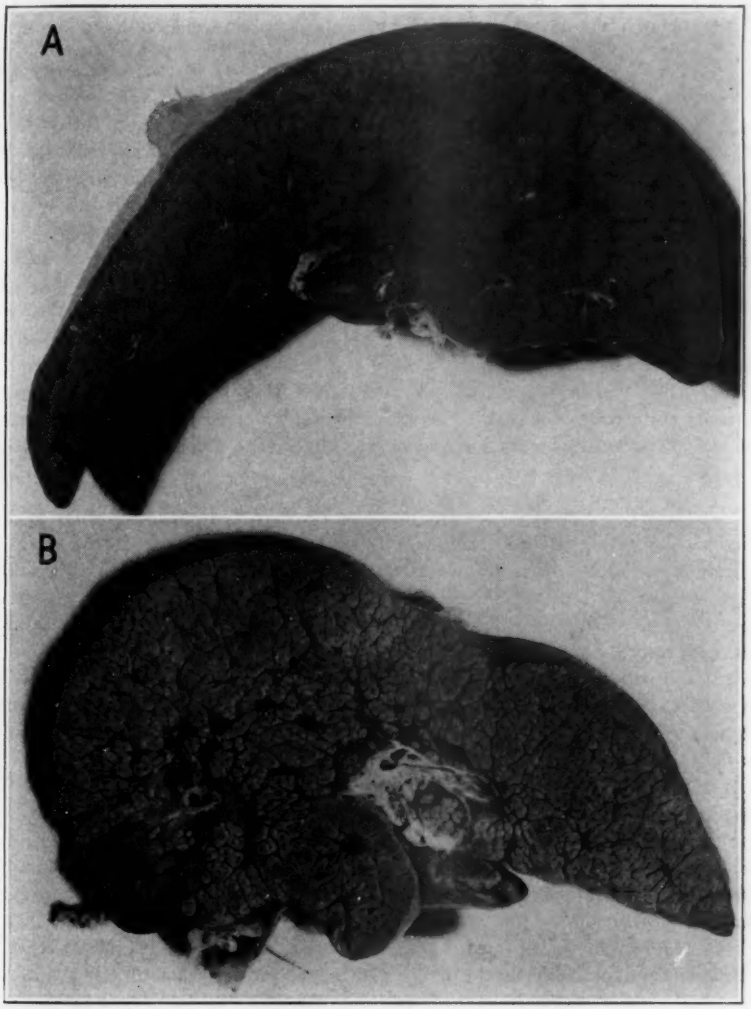

Fig. 6.—*A* (case 6), fine scarring resembling that caused by chronic infectious hepatitis in a patient 8 years old who died from typhoid fever. *B* (case 8), coarse scarring, with large regenerated nodules, in a patient 15 years old who had had repeated infections of the respiratory tract.

At autopsy, 800 cc. of straw-colored ascitic fluid, but no thrombosis, was found. The liver was composed of large yellow nodules (fig. 6 *B*). All lymphoid tissue was hyperplastic, and many organs showed petechiae. The regenerated

hepatic lobules had pushed the supporting framework to the periportal areas, where fibrosis, new bile ducts and occasional eosinophils were found. The spleen contained rare eosinophils. The serosa of the gallbladder contained lymphocytes and macrophages.

This patient probably had long-standing infectious hepatitis, with marked regeneration, terminating in thrombopenic purpura. The infection may have entered through the blood stream, secondarily involving the gallbladder.

CASE 9.—A 11 year old boy had had seven infections of the respiratory tract, with pneumonia and purulent otitis media, before the age of 5 years. Two years before admission he had had pharyngitis for one week; after three weeks abdominal swelling and hematemesis developed. Recovery was slow, and he never seemed well. Two weeks before admission, after two inoculations with typhoid vaccine, he had had hematemesis necessitating transfusions. He was pale and acutely ill, with an enlarged, tender abdomen. The tender spleen, which had an adherent, fibrous capsule and extended 5 cm. below the costal margin, was removed. The liver showed definite scarring. He is alive and free of symptoms after three years.

The capsule and trabeculae of the spleen were thickened by old fibrous tissue. Scattered through the fibrosed pulp were old hemorrhages and eosinophils.

The acute febrile onset followed by ascites and hematemesis suggested an infectious (or toxic) hepatitis. The exacerbation followed the injection of foreign protein. Since no specimen for biopsy was taken, the exact type of cirrhosis is unknown.

CASE 10.—Five months before death a 16 year old girl, admitted to City Memorial Hospital, Winston-Salem, N. C., had had a severe infection of the respiratory tract, followed by weakness, nausea and vomiting. Six days later jaundice, fever, abdominal tenderness and swelling, and pain in the midepigastrium and right flank developed, with generalized acne and pitting edema. Purple striae measuring 8 by 0.5 cm. appeared on the breasts and buttocks. Enlargement of the cervical and submaxillary lymph nodes, dulness and moist rales at the bases of the lungs were noted. Three weeks later, 2 quarts (2,000 cc.) of jaundiced ascitic fluid was removed, and nonmotile Escherichia coli, which did not form indole,[12] was cultured. She died after five months.

At autopsy, 2 liters of turbid ascitic fluid was removed. The liver was small and fibrous, with irregular yellow nodules measuring 6 mm. Petechiae were found on the omentum and serosa of the jejunum and ileum. Regenerated hepatic nodules were separated by masses of new and old connective tissue, containing numerous proliferating bile ducts and cells, lymphocytes and a few polymorphonuclear leukocytes.

This acute cirrhosis with marked regeneration was caused by infection of the peritoneum, and probably of the liver, with an atypical species of Escherichia.

Bacteria often are filtered out by the liver during septicemia or after metastasis through the portal system from pelvic and gastrointestinal or umbilical infections in the newborn. Inflammation, with destruction of hepatic tissue, develops into a portal type of cirrhosis during repair.

12. Bergey, D. H.; Breed, R. S.; Murray, E. G. D., and Hitchens, A. P.: Bergey's Manual of Determinative Bacteriology, ed. 5, Baltimore, Williams & Wilkins Company, 1939, p. 391.

Moon [13a] has described cases of the chronic, clinically mild type and MacMahon and Mallory [13b] the acute lesions in children due to beta hemolytic streptococci. Infection of the liver with Escherichia coli,[13c] Staphylococcus aureus [14] or Spirochaeta pallida [15] can produce an identical picture. We have seen no children in whom E. coli ascended the biliary passages to produce biliary cirrhosis.[13c] Both of these types have been reproduced in animals.[13]

CASE 11.—The first three pregnancies of the patient's mother had terminated in stillbirths. One week before admission an infection of the upper part of the respiratory tract had developed in this 7 week old Negro. He was given castor oil twice, which resulted in hematemesis and melena. Jaundice, nosebleeds and hemorrhages into the skin developed. He died after one hour.

At autopsy, the liver showed only jaundice. Pulmonary hemorrhages and widened lines of ossification of the bones were found. The hepatic lobules had retained their shape, but the cells were atrophied, and necrosis was scattered throughout. Fine fibrous tissue was increased throughout the lobules, especially in the periportal areas, where regeneration was beginning.

This patient had fine fibrosis in a liver damaged by congenital syphilitic infection. The acute terminal necrosis may have resulted from metabolic changes which followed the administration of castor oil.

CASE 12.—A 6 week old premature Negress had snuffles and for three days had regurgitated coffee-ground material after each breast feeding. She was undernourished and poorly developed, with pharyngitis, prominent abdominal veins and distention. The liver extended to the umbilicus, and the spleen extended 3 cm. below the costal margin. She died after twenty-four hours.

At autopsy, the abdomen contained 150 cc of clear fluid. The liver contained scattered yellow necrotic areas. The splenic capsule was thickened and the pulp fibrosed. The lungs contained firm white areas. The lobules were little distorted, but diffuse fine fibrosis had thinned the cords of hepatic cells. Abundant spirochetes were demonstrated. The splenic pulp contained scattered eosinophils.

This patient had diffuse hepatic fibrosis, with little necrosis, from congenital syphilitic infection.

CASE 13.—The third syphilitic child of an inadequately treated mother died after six hours. The liver extended below the umbilicus; the spleen was enlarged, and the palms and soles were scaling. At autopsy, a gumma measuring 5 cm. was found in the right lobe of the liver, with smaller scattered areas of necrosis and fibrosis (fig. 7). The lines of ossification of the bones were thickened and irregular. The hepatic lobules were diffusely infiltrated by fibrous tissue, which occasionally replaced hepatic cells except for isolated, thin, short cords (fig. 8).

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13. (*a*) Moon, V. H.: Infection as a Cause of Juvenile Cirrhosis, Am. J. M. Sc. **177**:681, 1929. (*b*) MacMahon, H. E., and Mallory, F. B.: Streptococcus Hepatitis, Am. J. Path. **7**:299, 1931. (*c*) MacMahon, H. E.: Infectious Cirrhosis, ibid. **7**:77, 1931.

14. Phillips, F. A.: Relapsing Staphylococcal Septicaemia Associated with Cirrhosis of the Liver and Splenomegaly, Lancet **1**:1050, 1937.

15. McMichael, J.: The Pathology of Hepatolienal Fibrosis, J. Path. & Bact. **39**:481, 1934.

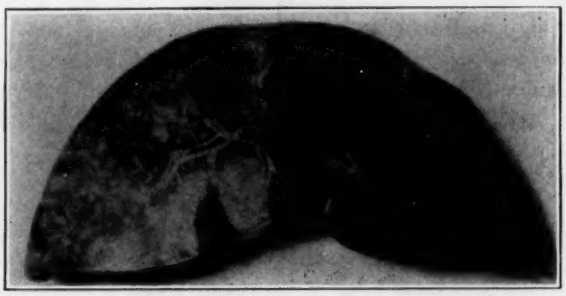

Fig. 7 (case 13).—Diffuse scarring, with gummas, in a newborn infant with congenital syphilis. Vessels with thickened walls and the normal gallbladder in cross section are seen at the left.

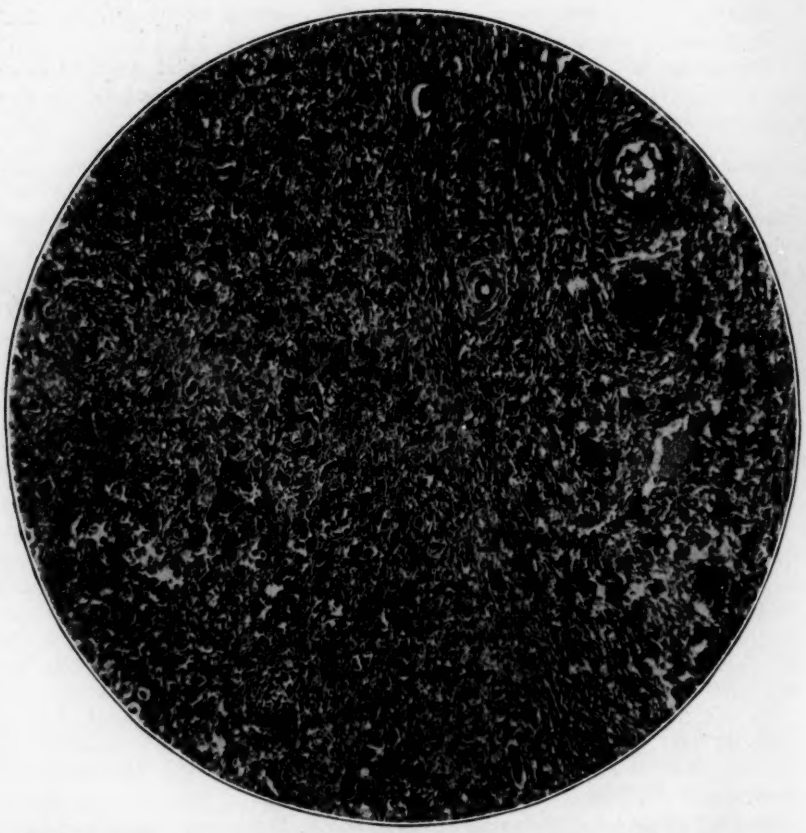

Fig. 8 (case 13).—Diffuse fibrosis in syphilitic infection in a newborn infant. A periportal area with concentric perivascular scarring is seen at the upper right; a linear scar is at the right center, and short, separated cords of hepatic cells are at the left center. Hematoxylin and eosin stain; × 90.

Scarring was pronounced around all blood vessels, particularly in the periportal spaces. Spirochetes were not demonstrated but were present in the preceding fetus.

This patient had diffuse and focal scarring caused by congenital syphilitic infection, which had progressed to formation of gummas in many places.

In the 4 patients with syphilis with cirrhosis, spirochetes were demonstrated in the liver only in patient 12, though the Wassermann test was positive in all. Anthelmintics were an additional toxic factor in patient 17, who alone showed the deep scars ascribed to syphilis; this may be due to the early age of the other patients. In all, diffuse lesions were present, with occasional localized gummas. No regeneration was

Fig. 9 (case 14).—Patient with cirrhosis following acute tonsillitis, with relief of ascites by omentopexy. *A*, taken Nov. 11, 1936, age 5 years, two months after onset, shows distention and scars of repeated paracenteses. *B*, taken June 2, 1938, shows operative scar and no ascites. *C*, infra-red photograph taken Aug. 24, 1939, shows dilated venous network three and a half years after operation.

nodular. Eighteen additional patients with congenital syphilis, some of whom were jaundiced, showed localized microscopic gummas and perivascular fibrosis or atrophy of the cords of cells but not cirrhosis.

Case 14.—Two months before admission tonsillitis developed in a 5 year old boy, and albumin, pus and casts were found in the urine. After three weeks anorexia, vomiting, jaundice, fever and melena were noted. The jaundice disappeared, but 5 quarts (5,000 cc.) of straw-colored ascitic fluid was removed. Enlargement of the cervical lymph nodes and prominent abdominal veins were

found. Omentopexy was done, and a specimen was taken for biopsy of the small, finely granular liver; the firm spleen was three times normal size.

The hepatic lobules were distorted, without recognizable central veins, and the cells were arranged in irregular groups separated by narrow bands of proliferating fibrous tissue.

He was last seen three years after operation, when only a dilated abdominal venous network was noted (fig. 9). Studies of hepatic function showed a reduction in the excretory function (table 1).

This patient's disease represented the healed stage of subacute hepatic necrosis, with subsequent obstruction to the portal circulation. A toxin originating from the tonsillitis was a possible etiologic factor; the nephritis was evidence of simultaneous renal damage.

The destruction of cells by bacterial toxins in the absence of demonstrable organisms in the liver and the subsequent development of cirrhosis have been described in human autopsies, but little confirmatory experimental work is available. Scarlet fever is usually incriminated, though diphtheria and measles are also mentioned (Moon,[13a] MacMahon and Mallory [13b]).

Case 15.—A 9 year old boy at Roanoke Rapids Hospital, Roanoke Rapids, N. C., had had rickets in infancy and malaria. One year before admission hookworm ova were found. Four weeks before admission gastric and intestinal hemorrhages, bleeding from mucous membranes and pain in the abdomen and back began. Splenectomy was done. He required repeated abdominal paracenteses and died one month later without autopsy.

The splenic pulp showed scattered eosinophils and lymphocytes, diffuse fibrosis and thickening of the trabeculae.

Though no section of the liver was made, the course and splenic findings were typical of cirrhosis. The cause was unknown; the malaria and hookworm may have been significant.

Fine, diffuse, multilobular cirrhosis was reported in Bombay [16] and Trinidad [17] as common in children, with enormous splenic enlargement and little ascites; the demonstration of malarial parasites preceded the finding of cirrhosis. Rogers,[18] from Calcutta, reported identical findings from kala-azar. In Calcutta in 1907,[19] 636 children under 2 years died from "biliary" cirrhosis with marked "intercellular fibrosis." The course and findings differed from those of malarial cirrhosis, but a parasitic agent was suspected.

The possible etiologic significance of intestinal parasites has received scant attention. Duprey [17] mentioned the presence of ascarides in 1 child (see our case 18), and Rogers [18] mentioned the frequency in India of

16. Tucker, E. F. G.: Malarial Cirrhosis of the Liver, Lancet **1**:1474, 1908.

17. Duprey, A. J. B.: Malarial Cirrhosis of the Liver, Lancet **2**:308, 1908.

18. Rogers, L.: The Aetiology of Cirrhosis of the Liver, Lancet **1**:895, 1911.

19. Pearse, T. F.: The Biliary Cirrhosis of Children, Otherwise Known as "Infantile Liver," Lancet **1**:165, 1909.

cirrhosis in adults with chronic amebic dysentery. The hookworm is said to liberate a toxin, but hepatic lesions following infection with hookworms are rarely described.

CASE 16.—A 4 month old girl was questionably jaundiced at birth, but after three weeks jaundice was progressive. At 2 months the stools became white and the urine dark. She was acutely ill, with pharyngitis, catarrhal otitis and pneumonia. The liver extended to the iliac crest with a sharp, smooth, firm edge; the spleen was barely palpable. At exploration, the bile ducts and gallbladder appeared normal. Fever increased, and she died three weeks postoperatively.

At autopsy, the liver was enlarged, firm and fibrous. Lymph nodes surrounding the common duct did not compress it; the gallbladder was thickened and collapsed. The lobular architecture remained, but marked necrosis was present in the central and middle zones, with collapse of the stroma and occasional hemorrhage. Old fibrous tissue infiltrated with a few mononuclear cells was found between the lobules in the periportal spaces. Rare new bile ducts and regenerated cells were present. Rare mononuclear cells were found in the mucosa and muscularis of the gallbladder, with minimal fibrosis of the villi. An interstitial mononuclear pneumonia was present.

This patient had acute necrosis in a slight chronic portal cirrhosis. The unknown original etiologic agent, in combination with the prolonged infection in the lungs, probably produced the old peripheral regeneration. The terminal necrosis in a previously damaged liver possibly was caused by anesthesia (ether).

CASE 17.—A 6 year old Negro had received "worm medicines" for years. One month before admission he became jaundiced and listless. Three weeks later he vomited and complained of dull pain over the liver. He was undernourished and distended, and the smooth, tender liver extended 3 cm. below the costal margin. No stigmas of congenital syphilis were present. Two weeks after admission profound shock, with a red blood cell count of 500,000, coincided with enlargement of the liver and development of ascites. He died three weeks later.

At autopsy, 2 liters of dark ascitic fluid was found. The surface of the liver was fissured; two areas of fine scarring measuring 5 cm. were present. An area of inflammation measuring 3 cm. was present in the cecum. The lobules were distorted by dense wide bands of old fibrous tissue, containing few lymphocytes, and new bile ducts which replaced hepatic cells or caused fine periportal, midzonal or central scarring.

This patient had fine and coarse hepatic scarring from the combination of congenital syphilitic infection and repeated doses of a poison, possibly carbon tetrachloride. The recent cecal infection may have metastasized terminally.

CASE 18.—A 6 year old girl at Saluda, N. C., had had "influenza" at 18 months, with a prolonged convalescence, and had never entirely recovered. She passed six round worms the day of examination. She was well developed, pale, drowsy and listless, with inflamed tonsils. The heart was enlarged, and a diastolic murmur was heard. Progressive jaundice began, and after one month she was desperately ill, with generalized edema and coarse rales in the chest. On removal of icteric ascitic fluid the liver was palpable 6 cm. below the costal margin. She died five weeks after onset.

At autopsy (Dr. E. McQueen Salley), the omentum was adherent to all contiguous tissues, and the pleural and pericardial spaces were obliterated by adhesions.

The hard liver and the pancreas were large and nodular. The lobular configuration was little distorted, but the central areas were necrotic, with the framework of connective tissue uncollapsed. In the periportal areas scattered lymphocytes and marked proliferation of bile ducts were found. Some canaliculi were distended with inspissated bile, but the large ducts were empty.

This patient had marked acute and chronic central necrosis, with early periportal regeneration, probably from a combination of passive congestion, infection of the respiratory tract and intestinal parasitism.

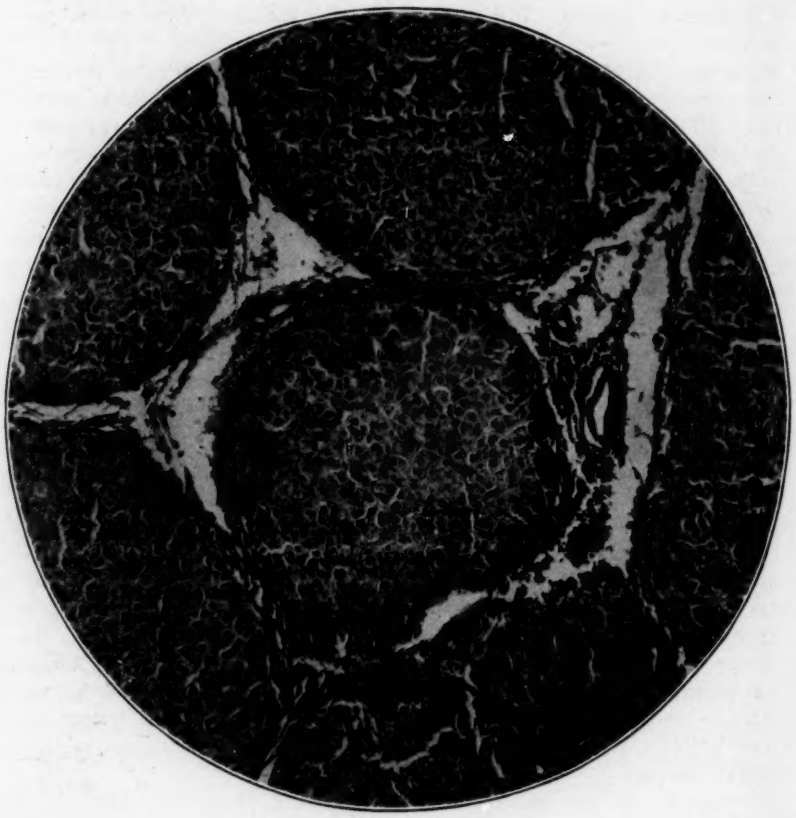

Fig. 10 (case 19).—Inactive cirrhosis in a 4 month old infant, with nodular regeneration and minimal scarring. A periportal area is seen at the upper right, with large thin-walled blood vessels separating the nodules. Hematoxylin and eosin stain; × 90.

Cirrhosis rarely can be traced to a single etiologic factor; the changes noted at autopsy result from the combined action of multiple agents. The classic production of cirrhosis by Opie,[20] who damaged the liver

20. Opie, E. L.: On the Relation of Combined Intoxication and Bacterial Infection to Necrosis of the Liver, Acute Yellow Atrophy and Cirrhosis, J. Exper. Med. **12**:367, 1910.

by chloroform or phosphorus and then injected E. coli or streptococci intravenously, has shown clearly that these lesions cannot be duplicated by either intoxication or infection alone (see case 2). Biliary cirrhosis usually is produced by a combination of obstruction and infection;[13] we have seen no examples in children. Diet is probably a factor in all cases.

Fig. 11 (case 19).—Patient with cirrhosis of unknown cause, the diagnosis being proved at the age of 4 months. *A*, lateral view taken Oct. 20, 1938, aged 5 years. *B*, infra-red photograph showing dilated venous network four and a half years after splenectomy.

CASE 19.—One week before admission a mass had been noted in the left side of a pale, poorly nourished 4 month old girl. The firm, sharp edge of the liver extended 6 cm. below the costal margin. The 8 cm. long spleen was removed, and a biopsy of a specimen from the scarred liver was done.

The liver was composed of regenerated nodules separated by thin-walled vessels (fig. 10). Isolated normal periportal areas, little fibrous tissue and no leukocytes were present.

She was last seen five and one-half years after splenectomy and appeared underdeveloped and poorly nourished, with a hemorrhagic diathesis. Many fine telangiectases were present over the body and the face. Infra-red photographs showed collateral venous circulation (fig. 11). Studies of hepatic function showed no abnormality (see table 1).

The inactive or healed portal cirrhosis may have resulted from a congenital anomaly of the lobules or blood supply or from intrauterine damage of unknown cause, comparable to yellow atrophy.

CASE 20.—A pale, 12 year old boy had had hematemesis and melena for four months. A smooth, firm spleen, which extended 7 cm. below the costal margin, was removed at another hospital one month later; gross cirrhosis but no thrombosis of the splenic vein was noted. He died suddenly one year later. Autopsy was not performed.

The splenic pulp showed diffuse fibrosis, lymphocytes and rare eosinophils, with widening of all trabeculae; a few lymphoid follicles showed recent hemorrhage.

This case, with gross cirrhosis of unknown cause, was untyped, since no specimen was taken for biopsy. The splenic changes and the course were consistent with Banti's syndrome.

Warthin [21] and McMichael [15] studied carefully instances of Banti's syndrome, with and without gross evidence of cirrhosis, and are convinced that endophlebitis in the portal venous system is the lesion at fault. McMichael concluded that the liver always is simultaneously involved but that the extent may be less than in the spleen and hence overlooked. Winternitz [4] has noted thromboses in the portal system following necrosis or infection in the liver (see case 1). Adequate microscopic descriptions or photomicrographs of the cirrhosis in Banti's syndrome are rare; it usually is said to be atrophic, interstitial or only mild diffuse scarring with leukocytic infiltration.

COMMENT

Incidence.—From June 1930 to July 1939, 2,500 autopsies have been done in Duke Hospital; 560 were on children under 15 years, exclusive of stillborn and premature infants. Of 57 patients with cirrhosis, 9 were under 16 years. Four autopsies were performed in other hospitals. In 4 cases the diagnosis was made by biopsy of the liver, while in 3 it was confirmed by operation with pathologic examination of the spleen only. Four patients died; autopsy was not performed. Three patients remain alive.

Of the 20 patients reported on, 13 were boys and 7 were girls; 13 were white and 7 were Negro children. Six patients were 4 months of age or under; 5 were from 5 months to 6 years of age and 9 were from 7 to 16 years of age at the time of operation or autopsy. The onset occurred after the age of 14 only in patient 10, the youngest patient with

21. Warthin, A. S.: The Relation of Thrombophlebitis of the Portal and Splenic Veins to Splenic Anemia and Banti's Disease, Internat. Clin. **4**:189, 1910.

acute infectious cirrhosis from whom we have recovered bacilli. The duration of symptoms ranged from three days to five years before admission. All of the patients were anemic. The white blood cell count ranged from 3,280 to 29,600. The percentage of eosinophils varied from 0 to 2 in 13 patients; in patient 16 it reached 8 before death. Plasma proteins were low usually with reversal of the albumin-globulin ratio; in the 2 surviving children on whom biopsies were performed, the proteins are now normal. None of the patients showed signs of sclerosis of the central nervous system; brain sections were available only from patients 12 and 13. Purpura eventually developed in 4 patients; 3 patients had epistaxis and 3 bleeding from the mucous membranes; 1 had intra-abdominal, 1 urinary and 10 gastrointestinal hemorrhages. Only patient 5 showed clubbing of the fingers. The laboratory and clinical data are summarized in table 1.

During the nine years in which 14 of the 20 cases reported were studied in Duke Hospital, 7,056 new patients were seen in the pediatric outpatient clinic, and 7,439 admissions [22] of patients under 15 years of age were made to the wards. At the Harriet Lane Home, Baltimore, from 1912 to 1932, 16 patients with cirrhosis were found among 80,000 children, an incidence of 0.02 per cent.[23] A review of 10,932 autopsies at the Johns Hopkins Hospital from 1890 to 1929 revealed 246 cases of cirrhosis, in 11, or 4.4 per cent, of which the patients were children under 15 years of age. In 4 of these 11 cases the condition was atrophic, in 3 biliary, in 2 circulatory and in 2 unclassified. In 3 cases it was due to atresia of the bile ducts, in 1 to phosphorus poisoning and in 2 to blood dyscrasias; 7 of the children were jaundiced, and 3 had ascites.[24] Of 9,346 autopsies at the Boston City Hospital from 1897 to 1932, 550 were done on patients with well marked cirrhosis of the liver. Of 10 patients in the first two decades of life, 7 had healed acute yellow atrophy, 3 were syphilitic and 1 was not classified.[25] In Great Britain, 40 cases of cirrhosis in 5,500 autopsies on children were found by Forbes [26] over a period of forty-five years, and 12 cases of portal cirrhosis in patients under the age of 21 years were found in 1,600 autopsies in the fifty-four years from 1865 to 1919.[27]

22. This figure includes readmissions of old patients. Most of those admitted had been previously studied in the outpatient clinic.

23. Arena, J. M., and Harris, R. R.: The Frequency and Distribution of Diseases in Children, South. Med. & Surg. **97**:520, 1935.

24. Robinson, E. C.: Unpublished data.

25. Mallory, F. B.: Cirrhosis of the Liver, New England J. Med. **206**:1231, 1932.

26. Forbes, J. G.: A Case of Tuberculosis of the Tonsils and Lymphatic Glands, Together with Congenital Bronchiectasis of Both Lungs and Cirrhosis of the Liver and Pancreas, Tr. Path. Soc. London **57**:347, 1906.

27. Rolleston, H., in Christian, H. A.: Oxford Medicine, New York, Oxford University Press, 1936, vol. 3, pt. 1, p. 384.

The protocols of all autopsies performed on children at Duke Hospital from June 1930 to July 1939 were examined. If hepatic lesions of any type were mentioned, microscopic slides of the liver, spleen and

TABLE 2.—*Pathologic Data for Twen*

			Gross Examination						
			Liver		Spleen				
Case	Operation	Autopsy	Weight, Gm.		Weight, Gm.		Gallbladder	Other Findings	Necrosis
1	..	+	280	Soft, nodular	80	Scarred capsule	Normal	Thrombus in portal vein	+++
2	..	+	640	Finely granular	130	Congested	Normal	Pneumonia, nephrosis	++
3	+	..	...	.....	...	.....	.....	.....	++
		+	950	Nodular	200	Normal	Normal	Pleurisy	++
4	+	..	...	Size 1½ × normal, extremely scarred	...	Size 2 × normal	Atrophied, fibrous	Other viscera normal	0
5	..	+	...	Enlarged, firm, scarred	...	Enlarged, firm	Normal	Rheumatic pancarditis	+
6	..	+	...	Finely nodular, scarred	150	Firm	Normal	Ulceration in colon	+
7	+	..	...	Size 2 × normal, granular	...	Normal	.....	.....	0
8	..	+	1,200	Nodular, scarred	350	Soft	Edematous	Lymphadenopathy, petechiae	0
9	+	..	...	Normal size, scarred	235	Scarred capsule	.....	.....	..
10	..	+	1,000	Nodular, fibrous	325	Normal	Normal	Hemorrhagic omentum	++
11	..	+	...	Size 2 × normal	...	Size 3 × normal, firm	Normal	Petechiae	+
12	..	+	225	Small necroses	50	Fibrosed	Adhesion to colon	Pneumonia, periostitis	+
13	..	+	210	Gumma in right lobe	45	Enlarged	Normal	Periostitis	++
14	+	..	...	Small, finely granular	...	Size 3 × normal, firm	.....	Other viscera normal	0
15	+	..	...	Normal size	200	Hard	.....	.....	..
16	..	+	...	Fibrous, normal size	20	Normal	Normal	Pneumonia	++
17	..	+	800	Fissures, large scars	250	Fibrous	Normal	Ulceration in cecum, bronchitis	+
18	..	+	...	Greatly enlarged, hard	...	Slightly enlarged	.....	Serous surfaces adherent	++
19	+	..	...	Nodular	...	Size 3 × normal, smooth	.....	.....	0
20	+	..	...	Finely scarred	325	Firm, adhesions	.....	No thrombus	..

gallbladder and the clinical record were reviewed. In each of the cases reported iron, bacterial, phloxine, scarlet red and Mallory connective tissue stains were done on sections of liver; iron and Mallory stains on sections of spleen, and bacterial stains on sections of gallbladder when available (table 2). Areas of necrosis were found in various parts of the hepatic lobules in numerous diseases, such as Flexner dysentery,

meningococcic (?) meningitis, purpura and eclampsia (in a 13 year old Negro girl), but these lesions were too acute to show evidence of scarring or regeneration. Localized areas of degeneration and scarring, with

atients with Cirrhosis of the Liver

Microscopic Examination													Classification			
Liver								Spleen			Gallbladder					
Necrosis	Fibrosis	Regeneration	Hyalin	Fat	Iron	Bacteria	Leukocytes	Fibrosis	Hemorrhage	Iron	Fibrosis	Leukocytes	Portal	Biliary Central		Unclassified
+++	++	++	+	++	0	0	+	..	..	..	..	..	+	..	..	..
++	+++	+++	++++	+++	0	0	++	++	++	+	0	0	+	..	..	..
++	+++	++++	++	..	0	0	+	..	..	..	..	..	..	..	..	..
+++	+++	++++	++++	++	0	0	+	++	++	0	0	0	+	..	..	..
0	+++	+++	0	0	0	0	+	..	..	..	..	..	..	+	..	..
+	++	0	0	++	++	0	0	+++	+	+	..	..	..	..	+	..
+	++	+++	±	+++	0	0	+++	0	++	0	0	0	..	+	..	..
0	++	+	0	+	0	0	+	..	..	..	..	..	..	..	..	+
0	+++	++++	0	0	0	0	+++	++	0	0	0	+	..	..	..	+
..	..	..	..	..	..	..	..	+++	+	0	..	..	..	..	..	..
++	+++	++++	±	+++	0	0	++	0	+	+	0	0	+	..	..	..
+	++	+	±	+++	+	0	+	+++	+	0	..	..	..	..	..	+
+	++	0	0	++	+	+++	+	+++	+	++	..	..	..	..	..	+
+++	+++	+	0	+	+	0	++++	+++	0	++	..	..	..	..	..	+
0	++	+++	+	..	0	0	+	..	..	..	..	..	+	..	..	..
..	..	..	..	..	..	..	..	++++	0	0	..	..	..	..	..	..
+++	+	+	0	+	+	0	+	+	0	++	+	0	+	..	..	..
+	+++	++	0	++	+++	0	++	++	++	+	0	0	..	..	..	+
++	+	++	0	++	++	0	+	+	0	0	..	..	..	+	..	..
0	+	++++	0	..	0	0	0	++++	0	+	..	..	+	..	..	..
..	..	..	..	..	..	..	..	++++	++	..	..	..	..	..	..	..

some evidence of regeneration, were found in instances of primary liver cell carcinoma and malignant tumor metastasized from the kidney. Marked fatty change or atrophy of the cords of cells after chloroform anesthesia and Vincent's angina and periportal scarring of unknown cause, without degeneration or regeneration, were noted. In addition to the instances of congenital syphilis already mentioned, 17 autopsies on

children showed lesions which in healing could have resulted in the microscopic picture of cirrhosis. Cases 1, 16 and 18 are instances of necrosis in which scarring and regeneration were sufficient to warrant the diagnosis of early cirrhosis.

Classification and Etiology.—A satisfactory classification of cirrhosis is impossible. Mallory could not classify 104 of 550 cases in patients of all ages.[25] We classified 7 instances as portal, 3 as biliary and 1 as circulatory, leaving 6 diffuse or unclassified, exclusive of the 3 instances of gross scarring without microscopic sections.

The usual classification is purely anatomic; this lacks meaning in cases 6 and 18, in which the patients' cirrhosis was microscopically biliary, though the gallbladder and bile ducts showed no lesions. Would these patients later have shown lesions in the gallbladder and biliary tract? Graham [28] expressed the belief that most biliary infections arise first as hepatitis; Noble [29] could not correlate the microscopic changes observed at autopsy in the periportal areas with those in the gallbladder. We have studied a 14 year old girl in whose case the microscopic observations suggested initial acute hepatitis with splenic fibrosis, followed by chronic periportal hepatitis, cholecystitis and cholelithiasis.

Gibson and Robertson [30] in reviewing 244 cases of obstructive jaundice found 21 in which the condition of the liver microscopically resembled Laennec's rather than biliary cirrhosis. We think Hanot's hypertrophic biliary cirrhosis is probably a phase of enlargement in the more common types; atrophy and increased scarring appear later.

An etiologic classification is acceptable clinically and pathologically, but a primary infectious etiologic agent other than S. pallida was demonstrated only in our case 10 and then not in hepatic tissue. No injurious chemical or metabolic agents were proved.

The importance of diet in preventing hepatic necrosis has been overlooked until recently. The fatty change in hepatic cells occurring in acute infections or debilitating diseases, such as infant diarrheas, long has been recognized, but the association with dietary deficiencies has not been stressed. Intrinsic errors of metabolism, diabetes and hyperthyroidism, are known to cause hepatic necrosis during acute exacerbations, when the metabolism of foods, chiefly carbohydrate, is upset. Connor [31] has recently shown that cirrhosis in persons with chronic alcoholism may result from the scarring which follows from fatty change in hepatic

28. Graham, E. A., and Peterman, M. G.: Further Observations on the Lymphatic Origin of Cholecystitis, Choledochitis and the Associated Pancreatitis, Arch. Surg. **4**:23 (Jan.) 1922.

29. Noble, J. F.: The Relation of Hepatitis to Cholecystitis, Am. J. Path. **9**: 473, 1933.

30. Gibson, W. R., and Robertson, H. E.: So-Called Biliary Cirrhosis, Arch. Path. **28**:37 (July) 1939.

cells deprived of normal tissue oxidation. The alcohol, a protoplasmic poison, is burned first at the expense of and not in place of sugar, with a resulting derangement of fat and carbohydrate metabolism. That the same process is at fault in diabetes has been shown in human autopsies and in experiments with depancreatized dogs and phlorhizin-treated guinea pigs.[31] Severe hepatic necrosis in thyrotoxicosis, in which atrophic cirrhosis may be a terminal result,[32] may be partially prevented by intravenous injections of dextrose before and after operation.

Fatty change in the liver, resembling the first stage of cirrhosis in alcoholism, occurred in children with diarrhea and vomiting; pathogenic bacteria were recovered rarely. Only a single acute episode occurred, and not the repeated insults to the liver seen in chronic alcoholism. In infancy and childhood, metabolism normally moves faster than in adults; the change is comparable to starvation when adequate food is not retained or absorbed. In many infants low carbon dioxide–combining power of the blood and acetonuria probably indicated insufficient combustion of carbohydrate, as well as loss of electrolytes. As autopsies of patients with histories of past diarrhea infrequently show hepatic lesions, the degenerative process is reversible if treated adequately and early. Repeated episodes may result in cirrhosis, especially if the partially damaged liver is infected. Patients 8 and 9 are striking examples of children who had repeated infections at distant sites and in whom cirrhosis subsequently developed. The dietary history of patient 8 was not available. The diet of patient 9 was deficient in vitamins, fresh vegetables and meat. The lack of detailed feeding histories prevents correlation with the hepatic lesions, but we think that dietary deficiencies are important in the development of cirrhosis and should receive attention, especially since they can be corrected parenterally.

Vitamin B_1 is essential to complete metabolism of dextrose and hence to hepatic function.[33] Degenerative changes may develop in hepatic cells [34] in animals fed excess carotene, provitamin A, in a limited diet A high fat diet protects dogs from acute yellow atrophy due to arsphenamine.[35] Andersen [36] found cirrhosis in 4 children with cystic fibrosis of the pancreas, in which fat metabolism is upset.

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31. Connor, C. L.: Fatty Infiltration of the Liver and the Development of Cirrhosis in Diabetes and Chronic Alcoholism, Am. J. Path. **14**:347, 1938.

32. Beaver, D. C., and Pemberton, J. deJ.: The Pathologic Anatomy of the Liver in Exophthalmic Goiter, Ann. Int. Med. **7**:687, 1933.

33. McCollum, E. V.; Orent-Keiles, E., and Day, H. G.: The Newer Knowledge of Nutrition, ed. 5, New York, The Macmillan Company, 1939, p. 465.

34. Hall, E. M., and MacKay, E. M.: Experimental Hepatic Pigmentation and Cirrhosis, Am. J. Path. **7**:343, 1931.

35. Craven, E. B., Jr.: The Importance of Diet in Preventing Acute Yellow Atrophy During Arsphenamine Treatment, Bull. Johns Hopkins Hosp. **48**:131, 1931.

36. Andersen, D. H.: Cystic Fibrosis of the Pancreas and Its Relation to Celiac Disease, Am. J. Dis. Child. **56**:344 (Aug.) 1938.

Little is known of the nervous control of hepatic cells or of the small blood vessels of the liver. The finding of focal cirrhosis, such as the "fatty infarct" or "lipoma," [37] or of the instance of isolated focal cirrhosis cited by MacMahon and Mallory [13b] is difficult to explain. We have observed a 17 year old Negress with an irrelevant past history who at autopsy had an unexplained inactive atrophic cirrhosis of the left lobe and a normal right lobe of the liver.

Pathogenesis.—The liver has great power of regeneration. In acute massive necrosis, yellow atrophy, if the patient survives regeneration may be complete. Because the damage occurs at a single time and regeneration proceeds before fibrosis is marked, little distortion may occur. If necrosis is scattered and repeatedly recurs over periods of time, a continuous process is set up of: (1) parenchymal degeneration with removal of the dead tissue; (2) parenchymal regeneration which often is nodular; (3) fibrosis. MacCallum [38] has concisely described this process for the portal type. Patches of cells are killed, and the remaining ones, partly disconnected from their bile ducts, regenerate, expand and compress the surrounding stroma on all sides. Another injury comes; these hyperplastic nodules are partially destroyed; new capillaries grow out from the intact cells, and the process is repeated until eventually no lobules remain but only hyperplastic nodules. The normal vascular tree in which smaller vessels come off at right angles is compressed and the vascular bed reduced and distorted.[39] The tension in the portal bed is increased and the stage set for the development of splenic fibrosis, ascites and collateral circulation. The hepatic blood supply is reduced, and further regeneration is retarded. Hepatic failure results from failure of the arterial blood supply to the parenchyma and not from the reduced amount of hepatic tissue present.[39]

The speed with which the final "crash" of hepatic insufficiency can progress is shown in patient 3, in whom necrosis of hepatic cells progressed markedly in the twelve hours between biopsy and autopsy. Perhaps the ether anesthesia was partly responsible,[40] as it may also have been in patients 4, 7 and 16, although ether did not cause clinical evidence (jaundice) of necrosis in patients 9, 14 and 19, who survived and in patient 15, who later died.

Palliative Measures.—No paracenteses have been necessary for patient 14 since extraperitoneal omentopexy was performed by the

37. Stewart, H. L.; Morgan, D. R., and Sprenkel, V. L.: Focal Fatty Change of the Liver and Focal Cirrohsis, Am. J. Clin. Path. **8**:405, 1938.

38. MacCallum, W. G.: Textbook of Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1932, p. 310.

39. McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis, Arch. Path. **5**:23 (Jan.) 1928.

40. Bourne, W.: Anesthetics and Liver Function, Am. J. Surg. **34**:486, 1936.

technic of Schiassi; our only patient treated thus, he is, we think, the youngest treated successfully.[41] The value of splenectomy is doubtful. Patient 19 in five and a half years and patient 9 in three years have progressed little since splenectomy, though studies of hepatic function have not been done of the latter child. In patient 14 the spleen was firm and three times normal size at operation (omentopexy), but progress has been equally satisfactory without splenectomy. In patients 15 and 20 and in instances of splenic anemia consistent with Banti's syndrome without gross evidence of cirrhosis, splenectomy did not alter the fatal course of the disease.

SUMMARY

Cirrhosis of the liver in children is more common than is usually suspected. The incidence reported clinically and in autopsies is reviewed. Microscopic slides and clinical records were reviewed in each instance of hepatic lesions of any type found in 560 autopsies on children performed over a period of nine years.

Twenty children with cirrhosis, of whom 13 came to autopsy, have been studied clinically and pathologically. The possible etiologic factors are discussed.

One child, proved by biopsy at 4 months of age to have cirrhosis of the liver, has survived five and a half years after splenectomy. Another child, who was proved by biopsy at 5 years of age to have cirrhosis and who is the youngest on whom a successful omentopexy has been reported, has survived three years. Though collateral circulation has been demonstrated in each, hepatic insufficiency is present only in 1, as evidenced by recent tests of hepatic function.

No satisfactory classification of cirrhosis has been proposed. The pathologic picture in children resembles that in adults, but classification is more difficult and often impossible.

The etiologic factors rarely are determined but are probably multiple. Diet deserves more consideration as a factor in the development of cirrhosis, especially when dietary deficiencies are combined with repeated infections.

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41. Levy, W.; Greenstein, N. M., and Leighton, B.: Portal Cirrhosis in Childhood, J. Pediat. **15**:91, 1939.

Obituaries

JOHN LOVETT MORSE, M.D.
1865—1940

Dr. John Lovett Morse died April 3, 1940, at his home in Newton, Mass. He was born in Taunton, Mass., April 21, 1865.

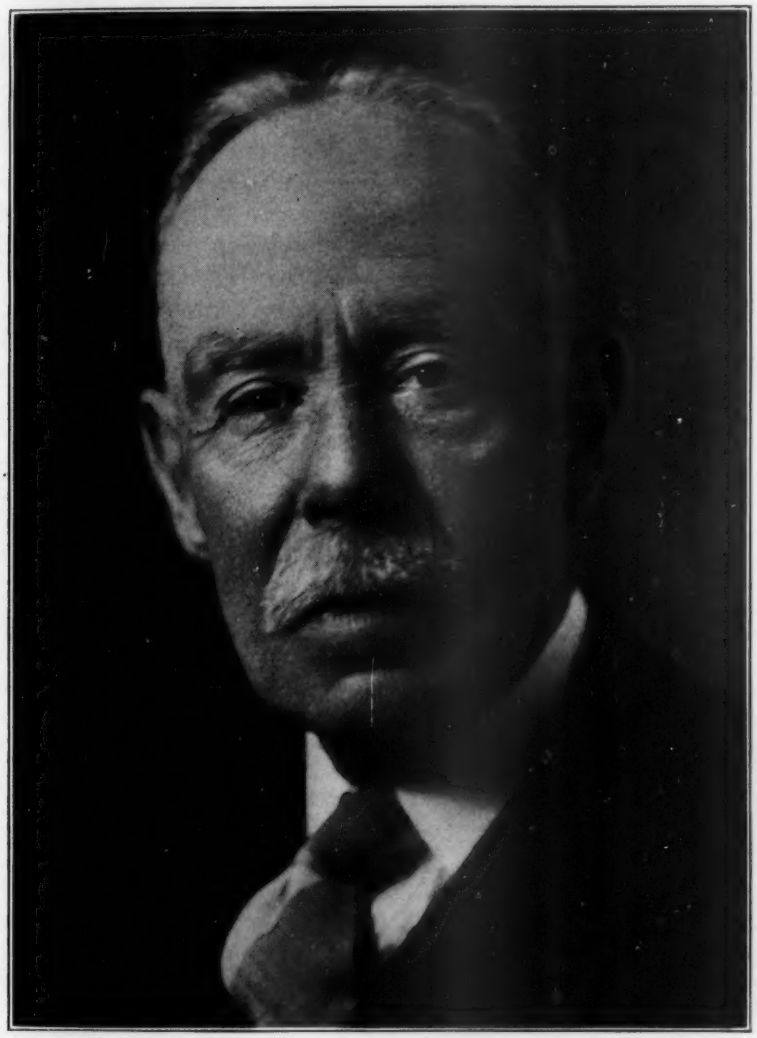
He graduated from Harvard College and Harvard Medical School, receiving the degrees of Master of Arts and Doctor of Medicine, in 1891. He served an internship at the Boston City Hospital and then for several years was connected with various other Boston hospitals—the Boston Dispensary, The Boston Lying-In Hospital and the Carney Hospital. He then began his practice in general internal medicine, and his first appointment at Harvard Medical School was as assistant in medicine, in 1896. Four years later he was transferred to the department of pediatrics. He rose rapidly through the various grades—instructor, assistant professor, associate professor—and in 1915 became professor of pediatrics. Before he devoted himself entirely to pediatrics he had worked in the orthopedic department of the Children's Hospital. During the term of his professorship in pediatrics he was chief of the medical staff at the Children's and Infants' Hospitals. He was also pediatric consultant to many hospitals in New England.

Dr. Morse was a member of the American Medical Association, the Massachusetts Medical Society, the American Pediatric Society and the American Academy of Pediatrics. He served a term as president of each of the latter societies and held the first certificate issued by the American Board of Pediatrics. He was an official examiner of the board up to the time of his death.

Dr. Morse wrote extensively on pediatric subjects. The articles were for the most part clinical. His chief interest was in the diagnosis and treatment of the individual sick child. He did, however, have a real interest in the supervision of the health of well babies and children and took an active part in the broader aspects of the work of the Academy of Pediatrics from the beginning. He was the author of two books, "Case Histories in Pediatrics" and "Clinical Pediatrics," and collaborated in the writing of two others, "Diseases of Nutrition and Infant Feeding," with Dr. F. L. Talbot, and "The Infant and Young Child," with Drs. E. T. Wyman and L. W. Hill.

Dr. Morse will be remembered primarily as an able clinician and as a forceful teacher of pediatrics. He excelled in demonstrations at the bedside of the patient. His observations were accurate, his thinking logical and his conclusions definite. He stated his ideas forcefully and

often with dry humor. He was painstaking in the application of all technics which might contribute to diagnosis. He called on his own experience and that of others, as recorded in the literature, in carrying out adequate therapeutic procedures.



JOHN LOVETT MORSE, M.D.
1865-1940

Dr. Morse was a hard worker and allowed himself little time for relaxation. His standards were high; he lived up to those standards himself and expected his associates to display the same quality of service.

To him, anything worth doing at all was worth doing well. He was a stern disciplinarian but never unkind or unjust. He was willing at all times to express his opinions and prejudices. One always knew where he stood; he never sailed under false colors. He was courageous in expressing his point of view and was never intimidated by opposition. He did not resort to indirect methods in the pursuit of his objectives.

After he retired from active pediatric practice and teaching, his interest in children did not cease. He attended medical meetings with considerable regularity and frequently contributed sagacious comments on papers which were read. His own contributions were a reflection of his point of view with reference to medicine as a whole. He was conservative in outlook, somewhat impatient with a too rapid acceptance of new ideas or procedures but willing to accept proved facts which shed new light on the physiology or pathology of children.

Dr. Morse's personality made a deep impression on a generation of pediatric students, and the influence he exerted on pediatric thought was great. His death removes another of the elder statesmen in pediatrics. He will be greatly missed by a host of friends.

R. M. S.

News and Comment

GENERAL NEWS

Lectures on Pediatrics in Arkansas.—In cooperation with the Arkansas State Board of Health and the Children's Bureau, Department of Labor, the Committee on Child Welfare has arranged for Dr. Jean V. Cooke, Associate Professor of Pediatrics, Washington University School of Medicine, to give a series of lectures on pediatrics in the state between May 20 and June 24, 1940.

DEATHS

Dr. Eugéne Apert, the well known Parisian pediatrician, died suddenly on February 10, at the age of 72.

Abstracts from Current Literature

Metabolism; Infant Feeding; Milk and Other Foods

OVER TWO THOUSAND ESTIMATIONS OF THE p_H OF REPRESENTATIVE FOODS. MILTON A. BRIDGES and MARGORIE R. MATTICE, Am. J. Digest. Dis. **6**:440 (Sept.) 1939.

This is a study of the p_H value of over four hundred representative foods. Brief comment is given to the clinical value of food reaction.

MORRISON, Savannah, Ga.

THE NUTRITIONAL AVAILABILITY OF IRON IN MOLASSES. ROBERT S. HARRIS, L. MALCOLM MOSHER and JOHN W. M. BUNKER, Am. J. Digest. Dis. **6**:459 (Sept.) 1939.

This study shows that molasses is a rich and inexpensive source of available iron. Male albino rats were used as the subjects in this experiment.

MORRISON, Savannah, Ga.

PROTEIN REQUIREMENTS OF INFANTS. JOHN ZABORSKY, Arch. Pediat. **56**:311 (May) 1939.

Too much protein does not produce the best results as to growth. Injury from milk frequently is manifested in young infants. Infants will not thrive satisfactorily on protein or even on powdered milk unless additional carbohydrates are given.

ORR, Buffalo.

DIETARY PROTEIN AND THE REGENERATION OF SERUM ALBUMIN. III. THE POTENCY VALUES OF EGG WHITE, BEEF LIVER AND GELATIN. A. A. WEECH and E. GOETTSCH, Bull. Johns Hopkins Hosp. **64**:425 (June) 1939.

With the method previously described for assaying the efficiency of food proteins in forming serum albumin the values for egg white, beef liver and gelatin are reported. The potency value of egg white is high, approaching that of beef serum; beef liver has an intermediate position, and gelatin has very little value.

LYTTLE, New York.

THE IMPORTANCE OF MILK OF LOW CURD TENSION IN INFANT FEEDING. MAURICE L. BLATT, Illinois M. J. **75**:545 (June) 1939.

After a study lasting eighteen months Blatt concludes that commercial pasteurized milk with a curd tension reduced to below 20 Gm. by proteolytic enzyme action is desirable for infant feeding, since it is well tolerated and digested and in addition is easily, economically and safely prepared. In its preparation the bacteriostatic value of handling a food product at a low temperature is utilized in contrast to the practice of making the milk soft curd by boiling and allowing to cool slowly.

BARBOUR, Peoria, Ill.

THE UTILIZATION OF THE CALCIUM OF MILK BY PRESCHOOL CHILDREN. GLADYS KINSMAN, DOROTHY SHELDON, ELIZABETH JENSEN, MARIE BERNDAS, JULIA OUTHOUSE and H. H. MITCHELL, J. Nutrition **17**:429 (May) 1939.

These investigators found that only one fifth to one fourth of the calcium of milk was utilized and retained by 11 preschool children (5 girls and 6 boys). The

average retention of calcium expressed as the percentage of intake was 20 per cent for girls and 18 per cent for boys. FREDEEN, Kansas City, Mo.

THE INFLUENCE OF DIFFERENT LEVELS OF MILK INTAKE ON THE ACID-BASE MINERAL BALANCE OF CHILDREN. HELEN A. HUNSCHER, FRANCES C. HUMMEL and ICIE G. MACY, J. Nutrition **17**:461 (May) 1939.

These investigators followed the acid-base balance of 7 healthy children 5 to 10 years of age over seven hundred and fifteen days during which they were consuming from 400 to 1,000 Gm. of milk daily. All the children stored both acid and base during the entire study, but the quantity stored was greater with the higher levels of milk in the diet.

FREDEEN, Kansas City, Mo.

THE UTILIZATION OF THE CALCIUM IN VARIOUS GREENS. MARY SPEIRS, J. Nutrition **17**:557 (June) 1939.

The calcium of turnip greens is about as available to young albino rats during the period of rapid growth and calcification as is that of milk. The calcium of tendergreens, collards and kale is slightly less available. The calcium of New Zealand spinach (high content of oxalate) is not only unavailable but reduces the available calcium of the milk in the diet. FREDEEN, Kansas City, Mo.

THE HEAT INCREMENTS OF DIETS BALANCED AND UNBALANCED WITH RESPECT TO PROTEIN. TOM S. HAMILTON, J. Nutrition **17**:583 (June) 1939.

Hamilton presented data and tables expressing the effects of increasing the protein in the diet of growing male rats on utilization of dietary energy. Dried, extracted whole egg was substituted for starch in the diet, increasing the protein from 4 to 54 per cent. He confirmed the general conception that the better balanced a diet is in satisfying an animal's requirements, the smaller will be its heat increment (specific dynamic effect) and the greater its net energy value. With every increase in the percentage of protein until 18 per cent was present, the heat increment of the diet decreased; it remained constant when the protein content of the diet was 18 to 30 per cent and increased again with a protein content of 42 per cent or more. An inverse relation between the heat increments and the growth-promoting abilities of the diet was noted. As the protein in the diet increased up to about 16 per cent the net energy value of the diet for growth plus maintenance increased, remaining constant throughout administration of the well balanced diets (16 to 30 per cent protein) and decreasing rapidly with addition of excess protein (to 30 per cent or more). The percentage of protein in the diet independent of the amount of food consumed did not affect the percentages of available energy used for basal expenditures or the total voluntary activity.

FREDEEN, Kansas City, Mo.

THE CALCIUM CONTENT OF WHITE BREAD. WILLIAM W. PROUTY and WILLIAM H. CATHCART, J. Nutrition **18**:217 (Sept.) 1939.

Prouty and Cathcart have analyzed the calcium content of white bread, using thirty-nine of the most popular commercial brands from thirty-three bakeries and twenty-seven states. The average percentage of calcium found was nearly three times that reported by previous investigators; this was due to the increased use of milk solids and yeast foods by commercial bakers. Assuming that the total amount of calcium is assimilable, six slices of average commercial white bread would supply approximately 30 per cent of the daily calcium requirement of the average adult. FREDEEN, Kansas City, Mo.

A Simplified Infant Feeding Formula: A Report of the Use of Irradiated Evaporated Milk and Water in 2,004 Cases. Henry O. McMahon, Wisconsin M. J. **38**:874 (Oct.) 1939.

A three year record of newborn infants was kept at the Milwaukee County Hospital. During the first year carbohydrates were routinely added to all formulas. During the second and third years, in an effort to reduce the digestive upsets, diarrheas and rashes so frequent among infants, standard mixtures of irradiated evaporated milk with no additional carbohydrate were tried. There were noted a striking reduction in the number of gastrointestinal upsets and a marked increase in the percentage of infants discharged from the hospital who were above birth weight. During the year in which carbohydrate was used, 38.4 per cent were discharged at or above birth weight, and during the "no sugar" regimen this percentage rose to 72.4 per cent. The standard milk mixtures also simplified hospital routine.

Janney, Wauwatosa, Wis.

Artificial Feeding with Mothers' Milk and Centers for Collection of Human Milk. R. Vaglio, Lattante **10**:237 (June) 1939.

Vaglio urges further increase in the number of centers for collecting and dispensing mothers' milk. A large city should have four or five small centers capable of furnishing 8,000 to 10,000 liters of milk per year.

Abstracter's Note: The Directroy for Mothers' Milk, Boston, collected 3,550 liters during 1938.

Higgins, Boston.

Is the Diet for the Second Year of Life Worthy of Special Study? M. Bergamini, Lattante **10**:275 (July) 1939.

The death rate in Italy for the first two years of life is higher than it should be. There are uncertainty, prejudices, mistakes and carelessness when direction of the feeding of the 1 year old child is left to the parents. The second year of life is important as to growth, mineral and vitamin intake and general habits and hygiene.

Therefore, the diet of the second year is worthy of being considered by itself and distinct from the diet of later years. An extensive discussion as to the proper diet follows, both the qualitative and the quantitative aspects being considered.

Higgins, Boston.

A New Biophysical Method of Differentiation Between Human and Animal Milk. M. Kasahara and S. Ogata, Ztschr. f. Kinderh. **61**:28 (June 6) 1939.

Breast milk shows considerable reduction of surface tension, and cow's and goat's milk show practically no reduction of surface tension after application of ultra sound waves.

Siemsen, Chicago.

Vitamins; Avitaminoses

Effects of Vitamin Deficiency on the Gastro-Intestinal Tract. D. L. Wilbur, Am. J. Digest Dis. **6**:610 (Nov.) 1939.

The gastrointestinal manifestations of vitamin deficiency include such symptoms as anorexia, diarrhea and abnormalities of function such as achlorhydria and alterations in the motor activity and in the absorptive function of the intestine. Also, lesions may be present, such as stomatitis, glossitis, proctitis or atrophy or inflammation of the gastric and intestinal mucosa. These manifestations are largely nonspecific.

Morrison, Savannah, Ga.

SIMULTANEOUS APPEARANCE OF A POSITIVE LINE TEST AND RADIOACTIVE PHOSPHATE DEPOSITION IN THE RACHITIC RAT METAPHYSIS. K. MORGAREIDGE and M. LEF. MANLY, J. Nutrition **18**:411 (Oct.) 1939.

This investigation deals with the artificially produced unstable radioactive isotope of phosphorus (P^{32}) as applied to studies of calcification. Morgareidge and Manly gave to rachitic rats a single dose of sodium phosphate, which contained the isotope (P^{32}), and a single dose of vitamin D_2. Then they determined the radioactive phosphorus content of whole blood and of the tibial diaphysis and metaphysis of each rat. Apparently vitamin D had no effect on the entrance of phosphorus into the blood or into the tibial diaphysis; in the metaphysis, however, the effect was definite (as shown by a positive line test). This effect was not noted until fifty-four or more hours after the administration of the vitamin. The authors' conclusion is that vitamin D has for at least one of its actions a direct influence on the metabolic factors controlling calcification, which is in contradistinction to the possible role it may play in intestinal absorption.

FREDEEN, Kansas City, Mo.

ROLE OF THE ANTIDERMATOSIS VITAMIN AND A NEW WATER-SOLUBLE GROWTH FACTOR IN THE NUTRITION OF THE MATURE FOWL. J. C. BAUERNFEIND and L. C. NORRIS, J. Nutrition **18**:579 (Dec.) 1939.

An experimental diet for the mature fowl, deficient in the antidermatosis vitamin, was developed by subjecting the cereal portion and liver extract to prolonged dry heat and the casein to special purification. This diet was adequate in vitamins A, B, B_4, B_6, D, E, G, K and the antiencephalomalacia factor. Nicotinic acid was found not needed in avian nutrition.

When this diet for hens was modified for chicks and supplemented with antidermatosis vitamin, it was deficient in another factor required for chick growth. This new factor was shown to be destroyed by the prolonged treatment with dry heat, stable in yeast to autoclaving, water soluble, adsorbable on fuller's earth and present in milk, liver, yeast and fresh green grass. Necessary for reproduction in the domestic fowl are both the new factor and the antidermatosis vitamin.

FREDEEN, Kansas City, Mo.

VITAMINS IN NERVOUS HEALTH AND DISEASE. H. WORTIS, New York State J. Med. **39**:1179 (June 15) 1939.

The importance of the various vitamins as related to neurologic conditions is reviewed.

AIKMAN, Rochester, N. Y.

CONTENT OF ASCORBIC ACID IN SOME FOODS. E. INGLESSI, Riv. di clin. pediat. **37**:436 (May) 1939.

Examination of the milk of nursing mothers of Greek nationality revealed that the ascorbic acid content varied from 1.5 to 4.75 mg. per hundred cubic centimeters. The ascorbic acid content of colostrum and of milk during the second week of nursing was much higher than subsequently. Cow's milk showed 3.52 mg. of ascorbic acid per hundred cubic centimeters during June and 3.34 mg. during September and October. Boiling milk resulted in a loss of 30 per cent of the ascorbic acid and pasteurization in a loss of about 10 per cent. Data are also given on the ascorbic acid content of lemons, oranges, apples and tomatoes at different times of the year. The author concludes that milk is insufficient to supply all the vitamin C needs of the infant and advises the use of fruits rich in vitamin C—i. e., lemons and oranges.

HIGGINS, Boston.

CAN THE INTRADERMAL REACTION OF ROTTER REVEAL THE DEGREE OF CONCENTRATION OF VITAMIN C IN THE BODY? B. CERA, Riv. di clin. pediat. **37**: 455 (May) 1939.

The test of Rotter involves an intradermal injection of 0.01 cc. of a 1/100 normal solution (2 mg. in 4.9 cc. of distilled water) of 2,6-dichlorophenolindophenol. This forms a small blue area with a diameter of 2 mm. The speed of decoloration of this area according to Rotter is directly proportional to the degree of concentration of vitamin C in the organism.

In normal guinea pigs this decoloration took place in from three to five minutes, but after the injection of 50 mg. of ascorbic acid intraperitoneally the time of the reaction had diminished in one hour to one quarter the normal figure, and twenty-four hours later the time was still one half of the normal. However, in guinea pigs showing signs of active scurvy the time of disappearance of the color was the same as in the normal, and when ascorbic acid was given intraperitoneally to guinea pigs with scurvy there was no diminution in the rate of disappearance of the color. Since the test of Rotter is not applicable when there is a lack of vitamin C, substances other than ascorbic acid probably vitiate the value of the test.

HIGGINS, Boston.

EVALUATIONS OF VITAMINS A, B_1 AND C AS THERAPEUTIC AGENTS. S. KUTHAN, Wien. klin. Wchnschr. **25**:597 (June 23) 1939.

If investigators wish to evaluate the therapeutic effectiveness of vitamins A, B_1 and C, they cannot help but be disappointed occasionally if the use of these vitamins is not strictly confined to conditions for which they are specific, according to Stephen Kuthan. By the use of vitamin C, he obtained fairly good results in treating hemorrhagic stomatitis and gingivitis accompanying various diseases. He also obtained favorable results in treating the hemorrhagic diathesis in certain cases. He states that other therapeutic indications for this vitamin are questionable.

He obtained good results by using vitamin B_1 in cases of alcoholic neuritis and in neuritis accompanying gastrointestinal disturbances, but in treating other conditions the results with vitamin B_1 were questionable.

Vitamin A proved most valuable in those conditions in which a disturbance in the mucosal epithelium was evident.

He states that he does not wish to minimize the therapeutic value of these vitamins, and he also points out that to the aforementioned specific conditions one must add the various latent hypovitaminoses. In other words, a deficiency in the intake of the aforementioned vitamins, although not giving rise to clinical manifestations of disease, may play a part in the production of certain disease conditions. The question of resistance to avitaminosis as produced by Georges and Mouriquant has not yet been fully investigated. He emphasizes the fact that in certain conditions therapy with vitamin A, B_1 and C must be augmented by other indicated therapeutic agents.

ABT, Chicago.

FLUCTUATIONS IN THE VITAMIN C CONTENT OF THE BLOOD IN HEPATIC INJURY. F. KOSAKI, Acta pædiat. japon. **45**:640 (May) 1939.

When injury of the liver was produced by administration of chloroform, yellow phosphorus and carbon tetrachloride, the vitamin C content of the blood diminished rapidly, so that on the third day of intoxication the lowest level was reached (a 60 to 70 per cent decrease). The recovery was seen in ten to fourteen days. If vitamin C was given before hepatic damage was produced, the extent of the diminution in the vitamin C content of the blood was not altered, but the restoration of the normal level of this vitamin was hastened.

KATO, Chicago.

Hygiene; Growth and Nutrition; Public Health

GROWTH OF CHILDREN OF PRESCHOOL AGE IN BATAVIA. T. E. DHONG, R. SOEKONTO and J. H. DE HAAS, Indian J. Pediat. **6**:163 (July) 1939.

The native and Chinese children of preschool age in Batavia are in weight from one-half year to two years and in height up to one and one-half year behind the children of the same age in Glasgow, Peiping and the United States.

GONCE, Madison, Wis.

THE CHARLES MACKAY LECTURE. SIR ROBERT WADE, M. J. Australia **1**:371 (March 11) 1939.

Preschool children, that is, children aged from 2 to 7 years, are the ones most neglected. There is no organized supervision for them. They are left entirely to nursemaids and parents. The parents, by virtue of their parenthood, are supposed to know all about their job. What nursemaids are supposed to know the author says he has no idea. Up to 2 years of age children undergo supervision by "child health centers," but the gap from there to the time the child enters school needs considerable attention. "There should be a series of nursery schools scattered throughout the state, which would have to be maintained by the state government. There should also be in each state one or more model and research training schools which, in addition to doing the work for the children who attend it, would act as a training school for teachers and heads of such nursery schools." The nursery school should be in close contact with a children's hospital and with "child guidance clinics." A nutritional investigation bureau attached to the children's hospital should supervise and study the nutrition of the pupils.

Many medical problems exist in the preschool age, such as the results of malnutrition in infancy as they affect later nutrition and dentition and the effect of nutrition on skeletal growth. Studies on infectious disease are of great importance, and some uniform method of statistical studies must be developed. Studies of infections of the upper respiratory tract and of infantile paralysis are important. Problems of mental hygiene are of the utmost importance during this period. It is the great rate of mental development in the first five years that necessitates such careful training in behavior, and the mother, with her multitudinous other duties, is frequently unable to meet the situations with the unruffled front so necessary to smooth development for the child.

It must be recognized that there is still no exact knowledge as to the best methods of training and that the swing from parental autocracy to extreme liberalism is still going on.

Continued research in nutrition is necessary to determine the real role of the endocrine glands, of methods of cooking and of the importance of various minerals and vitamins in the complete picture of child health.

TENNEY, Madison, Wis.

PHYSICAL EDUCATION IN THE DEVELOPMENT OF THE YOUNG GIRL. G. DREYFUS-SÉE, Rev. méd. sociale et de protec. de l'enf. **7**:81 (March-April) 1939.

For the period preceding puberty the author recommends breathing exercises and exercises for correction of habitually bad posture and for development of the abdominal and dorsal muscles. He advises that gymnastics be continued after the beginning of menstruation, but certain precautions are necessary to prevent interference with the development of the reproductive organs. He gives descriptions of special exercises. For girls with menstrual troubles the author advises therapeutic gymnastics under the strict supervision of a physician. The conclusion is that gymnastics for girls should concentrate on harmonious development of the body, improvement of posture and preparation of the girl for motherhood by promotion of normal functioning of her reproductive organs.

BIERMAN, Washington, D. C.

REPLY TO THE QUESTIONNAIRE ON THE PROBLEM OF INFANT MORTALITY DURING THE FIRST TWO YEARS OF LIFE. M. BERGAMINI, Lattante **10**:131 (March) 1939.

The hope of reducing the infant mortality of immature, syphilitic and rachitic children involves organized pediatric and nursing services under state direction. To reduce the number of deaths from nutritional disturbances, the dispensing of infant feedings to certain mothers by community health centers is suggested. To reduce mortality from infections of the respiratory tract and contagious diseases, better prophylactic measures and isolation are necessary. The use of convalescent serum in measles, vaccine in whooping cough, more universal vaccination in diphtheria and removal of the infant from the source of infection in tuberculosis seem to offer promise of a reduced number of deaths. To diminish infant mortality among the illegitimate, the inauguration of central supervision of orphanages and of other services for the illegitimate is recommended. HIGGINS, Boston.

REPLY TO THE QUESTIONNAIRE ON THE PROBLEM OF INFANT MORTALITY DURING THE FIRST TWO YEARS OF LIFE. R. CAPORALI, Lattante **10**:135 (March) 1939.

Caporali reports that infant mortality is highest in the mountain communities, next in industrial communities, next in farming communities and lowest in lake and sea coast communities. The mountain communities are characterized by lower economic resources and standards of living, poorer hygiene, less available medical care and less alert mentalities. In the industrial communities the tendency of the mothers to work and to leave the children at home results in poor care for the children and a higher death rate; gastroenteritis is frequent in these communities. In the agricultural communities some of the mothers work, but the incidence of breast feeding is much higher than in the industrial communities. The lowest death rate is in the coastal and lake districts, where the standard of living is higher, education and hygiene are better and contact with foreigners is greater.

HIGGINS, Boston.

WHAT IS INCLUDED UNDER PUERICULTURE? R. VAGLIO, Lattante **10**:153 (April) 1939.

By recent legislative decree the teaching of puericulture has been made compulsory in the women's high schools and in the universities. In the University of Naples there has been established a School of Puericulture to meet this requirement.

The questions arise what is the scope of puericulture, and how shall it be taught? Certainly a more advanced course is necessary in a university than in a high school. Puericulture, in brief, points toward elimination of poor heredity, guarantee of the best possible body and avoidance of the deleterious effects of environment. A course should include the study of housing, climate and general prophylaxis against disease. The institute of puericulture should afford a field for specialization both for nurses and for physicians. Puericulture, according to modern Fascist ideas, will expand the field of the medical student and the physician so that he or she is not only a clinician and a therapeutist but a hygienist and an apostle. HIGGINS, Boston.

CENTERS FOR COLLECTION OF MOTHERS' MILK. G. CRISTALLI, Lattante **10**:357 (Aug.) 1939.

Cristalli defends himself against a charge of hyperenthusiasm for the collection of mothers' milk to meet the nutritional problems of infants. HIGGINS, Boston.

PLAN OF "INFECTIOUS DISEASE WARD" PERMITTING VISITORS. REYNOLDO AGRELO and F. ARRISTIA, Rev. méd. latino-Am. **24**:709 (April) 1939.

Ten beds are placed in a ward, five in a row, head to head, with the heads in the center of the room. They are separated by glass partitions. A railing is

placed around the edge of the room, forming a visitors' aisle which permits parents to see the patient and talk to the nurse at a safe distance. Two sketches accompany the article.

DWYER, Kansas City, Mo.

Prematurity and Congenital Deformities

CASE REPORT OF A PREMATURE BABY. LOUIS FISCHER, Arch. Pediat. **56**:50 (Jan.) 1939.

A case is reported.

ORR, Buffalo, N. Y.

WATER EXCHANGE OF PREMATURE INFANTS—COMPARISON OF METABOLIC (ORGANIC) AND ELECTROLYTE (INORGANIC) METHODS OF MEASUREMENT. HARRY M. GORDON, SAMUEL Z. LEVINE, ELEANOR MARPLES, HELEN MCNAMARA and HELEN R. BENJAMIN, J. Clin. Investigation **18**:187 (March) 1939.

Eight observations of water and electrolyte balance through periods totaling forty-eight days were made on 4 premature boys. The water balance was calculated from the metabolic mixture (estimates of the protein, fat and carbohydrate oxidized) and from the electrolyte exchange. In 5 of 8 observations the close agreement between the results of the two methods suggests that under carefully controlled environmental and dietary conditions either method of predicting the water balance of thriving premature infants is satisfactory. GUEST, Cincinnati.

STUDIES IN FETAL BEHAVIOR: I. FETAL HEART RATE AS A BEHAVIORAL INDICATOR. L. W. SONTAG and T. W. RICHARDS, Monograph of the Society for Research in Child Development, Washington, D. C., National Research Council, 1938, vol. 3, no. 4.

This monograph contains a report of the efforts of the authors to exploit the quantitative differences and changes in the fetal heart beat and rate of beat as two of the few indicators of physical change and response to stimuli in the human fetus. A discussion of technics is presented. A considerable body of data is devoted to the determination of the characteristics of the heart rate at various fetal ages, the degree of its variation in the normal fetus, particularly for very short time spans, since it is evident that a large number of short samplings will demonstrate types of variation very different from those demonstrated by a few long spans in which second by second changes would be lost. The developmental aspect of the heart rate is emphasized throughout, since rapid changes in the mechanism for nervous control of the heart are to be expected during the fetal period. Specific relationships which have been investigated are those between fetal heart rate and the condition of the maternal heart, fetal heart rate and maternal sleep, fetal heart rate and maternal meals, fetal heart rate and maternal inhalation of amyl nitrate, fetal heart rate and administration of oxygen to the mother, and maternal smoking and vibratory stimulation. A study of the modifiability of the fetal heart responses to vibratory stimulation by simultaneous maternal smoking is also included. The general conclusions are (1) that changes in fetal heart rate as responses to various stimuli are valuable indicators of fetal reactivity to such stimuli and (2) that a number of the stimuli investigated are perceived and responded to by the fetus.

PALMER, Washington, D. C.

CONGENITAL VESICO-VAGINAL FISTULA WITH IMPERFORATE HYMEN; HYDROPS FOETALIS AND ERYTHROBLASTOSIS: POLYDACTYLY. F. PARKES WEBER and M. SCHOLTZ, Brit. J. Child. Dis. **36**:131 (April-June) 1939.

An infant who died shortly after birth showed a large abdomen due to great distention of the cavity of the uterus and vagina with urine. The title lists the other findings.

LANGMANN, New York.

A Description of a Monster; Diprosopus Tetrophthalmus. I. Brodsky, Brit. J. Ophth. **23**:250 (April) 1939.

To the ophthalmologist the features of particular interest pertaining to the monstrosity described here concern the eyes. The bulgings above two noses contained eyes. The eyes and the palpebral margins of the lateral eyes appeared normal, notwithstanding an epicanthal fold of the inner angle. Inspection of the central bulge removed the impression that there was a single median eye. The palpebral margins provided a clue, in that a cavity was present almost in the middle of the inferior lid, though no ridges or depressions marked the conjunctival surface. Eccentrically placed and slightly to the left of the midline was an iris, approximately equal in size to the lateral irides. Retraction of the eyelids revealed another iris in the upper right quadrant. The corneas over these two irides were continuous, the corneosclerotic junctions taking the form of an hourglass. An illustration shows this well and, in addition, reveals strabismus, the axes of the eyes making an angle of 20 degrees in front.

A block containing the eyes exhibited maximum peripheral fusion at the corneal area. In a sagittal plane a moderately deep annular groove extended around the junction of the eyes and became flattened near the cornea. A common lateral rectus muscle served both eyes, although it was attached principally to the smaller eye, which incidentally received the larger of the two optic nerves. More than half of the cornea of the smaller eye was buried in the fusion, whereas the limbus of the larger eye was just covered.

Transition from sclera to cornea was clearly defined in the larger eye, being demarcated by large vessels. When the sclera was traced back it was found to become thin and compact at the junction of the two eyes. The large vessels at the junction probably indicated the anterior limit of the common sclera; over the front of this the cornea was continuous. Bowman's membrane extended from the extremity of one cornea to the outer edge of the other cornea and crossed the point of junction, where it was less well defined. In the smaller eye, deep to Bowman's membranes, were vessels which did not extend as far as the junction of the two corneas. The substantia propria of the smaller eye lost its lamination at the point where the two corneas were incompletely separated. Descemet's membrane and the endothelium of the smaller eye appeared to end abruptly at the junction of the substantia propria and the sclera. From this point to the angle the anterior chamber was lined by a thin membrane. Posteriorly, the common sclera separated into two parts corresponding to each eye. The vessels of the choroidal coats were large and full, but there was no abnormality of the retina. A wide gap intervened between the two optic nerves.

W. Zentmayer. [Arch. Ophth.]

Bronchiectasis and Visceral Transposition: Report of a Case. D. B. Rosenthal, M. J. Australia **1**:761 (May 20) 1939.

A case of complete transposition of viscera with bronchiectasis and sinal infection is reported. The association of these conditions is discussed, with brief reference to their etiology.

Gonce, Madison, Wis.

Dwarfism of Uncertain Causation. F. Parkes Weber, Proc. Roy. Soc. Med. **32**:608 (April) 1939.

The patient, a boy aged 13 years, was brought to the hospital on account of his small size in June 1938. He had weighed 7½ pounds (3,402 Gm.) at birth. He was an only child. The osseous, mental and sexual development corresponded with the age. On roentgen examination the pituitary fossa appeared small and the bridge of the nose was somewhat depressed. By November 1938, without special treatment, his size had increased. The Wassermann reaction was negative.

Williamson, New Orleans.

CONGENITAL ANOMALY OF THE SO-CALLED AMSTERDAM TYPE IN ITALY. BRUNO PINCHERLE, Arch. de méd. d. enf. **42**:443 (July) 1939.

The first case observed in Italy of a new form of degeneration, originally described in 1933 by de Lange as typus Amstelodamensis, is reported. Since de Lange's publication 4 further cases have been studied, all originating in Amsterdam. The fundamental characteristics of the congenital anomaly as fixed by investigators in Holland are: mental debility; great constitutional weakness; underweight; brachycephaly; hirsutism of the forehead; hypertrophy of the eyebrows and eyelashes; abnormally low setting of the ears; small hands and feet; proximal position of the thumb and thenar eminence; hook shape of the little finger, and partial bilateral syndactylism of the toes. The patients resemble each other in every detail, as in mongolism. The cause is obscure but undoubtedly concerns the germ plasm. In the Italian case there were also found persistent capillary immaturity and mixed astigmatism.

AMESSE, Denver.

SIAMESE TWINS. P. ANOKHINE, Presse méd. **47**:379 (March 11) 1939.

Anokhine presents a case of twins fused at the thorax. A common trunk supported two heads and four arms. Only two breasts were present. Roentgenograms revealed the presence of two hearts, two vertebral columns and two stomachs.

DAFFINEE, Boston.

Acute Contagious Diseases

THE USE OF SULFAPYRIDINE IN THE TREATMENT OF PNEUMONIA COMPLICATING PERTUSSIS. LEO LITTER, ABRAHAM M. LITVAK AND THURMAN B. GIVAN, Arch. Pediat. **56**:518 (Aug.) 1939.

Twenty-two cases of pertussis pneumonia were treated with sulfapyridine, with a favorable response in most instances.

ORR, Buffalo.

RHINOVACCINATION AND RHINOREVACCINATION. DESIDER GYURE, Arch. Pediat. **56**:529 (Aug.) 1939.

Twenty-eight patients were vaccinated through the uninjured nasal mucous membrane (rhinovaccination) and 62 patients were revaccinated by the same method, without a severe or a fatal reaction.

Although rhinovaccination, which might be considered to offer a physiologic way of absorption of the virus, gives at least the same result as cutaneous inoculation, preference is given to cutaneous vaccination for the first vaccination because the permanent scar serves as evidence.

ORR, Buffalo.

SULFANILAMIDE TREATMENT OF ERYSIPELAS. JOHN NELSON, HARVEY RINZLER and M. P. KELSEY, J. A. M. A. **112**:1044 (March 18) 1939.

Correlation of mortality rates with various methods of treatment reveals a pronounced decrease in mortality from erysipelas since sulfanilamide has been used. For children this figure was 37.5 per cent with antitoxin, 19.7 per cent with ultraviolet radiation and other methods and 12.9 per cent with sulfanilamide. The doses and reactions are described.

HEERSMA, Kalamazoo, Mich.

IMMUNOLOGIC METHODS IN PEDIATRICS. ARCHIBALD L. HOYNE, J. A. M. A. **112**:1581 (April 22) 1939.

Hoyne discusses the procedures which he advocates for immunization against the common contagious diseases, the methods of producing passive immunity and

the human and animal serum treatments for contagious diseases. The prophylactic and therapeutic doses, the methods of administration and reaction and the diseases in which these measures are indicated are described.

HEERSMA, Kalamazoo, Mich.

DIPHTHERIA IMMUNITY IN CHICAGO. HERMAN N. BUNDESEN, WILLIAM I. FISHBEIN and JOHN L. WHITE, J. A. M. A. **112**:1919 (May 13) 1939.

Comparison of the various types of immunization procedures for diphtheria by means of Schick tests and serum antitoxin determination revealed the superiority of multiple injections over one or two injections. The authors obtained their best results with three monthly doses of plain toxoid. The Schick test is not an infallible index, as was shown by the antitoxin value. Natural immunity and that acquired from exposure to carriers may enter into the results of immunization procedures.

HEERSMA, Kalamazoo, Mich.

ANTERIOR POLIOMYELITIS TREATED WITH SULFAPYRIDINE. JOHN C. WAGNER, J. A. M. A. **112**:1934 (May 13) 1939.

In the case of a 9 year old girl sulfapyridine given during the acute stages of poliomyelitis appeared to produce a drop in the temperature, a reduction in the leukocyte count and relief of the paresthesias. The paralysis was unchanged.

HEERSMA, Kalamazoo, Mich.

SULFANILAMIDE IN THE TREATMENT OF SMALLPOX. WALTER O. MCCAMMON, J. A. M. A. **112**:1936 (May 13) 1939.

After known exposure to smallpox 4 of 7 persons were treated with sulfanilamide on appearance of the symptoms. The other 3 passed through the usual stage, but in the 4 treated the severity of the eruptive stage seemed to be aborted. In only 1 did pustulation (very slight) occur.

HEERSMA, Kalamazoo, Mich.

TREATMENT OF EARLY MEASLES WITH PARENTAL WHOLE BLOOD: EFFECT ON CLINICAL COURSE AND PULMONARY COMPLICATIONS IN YOUNG CHILDREN. JEROME L. KOHN, ALFRED E. FISCHER AND HENRY ULMER RESCH, J. Pediat. **14**:502 (April) 1939.

In an attempt to reduce the incidence of pulmonary complications and thus lower the mortality, 66 children under 3 years of age in the preeruptive or early eruptive stage of measles were given subcutaneous injections of 20 to 40 cc. of adult blood. There was little difference in the severity of the measles or in the incidence of pulmonary involvement when these 66 children were compared with 758 children in the same age group who did not receive injections of blood.

RAUH, Cincinnati. (From Authors' Summary)

POLIOMYELITIS IN CHARLESTON IN 1939. J. I. WARING, J. South Carolina M. A. **35**:156 (June) 1939.

This is probably a preliminary report; it was given as of May 1939, at which time there had been 90 cases of poliomyelitis reported in Charleston County. Most of the patients were of preschool age. Undoubtedly a good many "abortive cases" were overlooked, including cases of minor paralysis. The writer lists the common symptoms of this epidemic: (1) slight congestion of the throat; (2) fever; (3) headache; (4) drowsiness; (5) vomiting; (6) stiffness of the neck, and (7) photophobia. The paralysis develops from three to seven days after onset. Diagnostic lumbar puncture was done in a number of cases. No specific treatment is advised.

WESTON, Columbia, S. C.

PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES. Pub. Health Rep. **54**:1195 (July 7) 1939.

For the period from May 21 to July 7, 1939, poliomyelitis was above the seasonal incidence, with 217 reported cases. Three fourths of these were observed in South Carolina, Georgia, Arizona and California. In other regions the number of cases was below the seasonal incidence. Smallpox was increasing, particularly in the Western and North Central Regions (1,057 cases). Influenza, although declining, was still above the median. Diphtheria, measles, scarlet fever, meningitis and typhoid fever were below the average. The average mortality was 10.6 per 1,000, which is the lowest for this period since 1933. SANFORD, Chicago.

ORTHOPEDIC CARE OF CONVALESCENT POLIOMYELITIS. W. VERNON NEWMAN, South. M. J. **32**:900 (Sept.) 1939.

The author believes that absolute rest until maximum nerve supply has returned is of paramount importance.

Great care should be taken to build up the nutritional state of the patient.

Braces to permit underwater exercises are used after 50 per cent function is demonstrable. They are also used to permit weight bearing and prevent deformities which casts fail to control.

Heat, massage, passive motion and electrical stimulation of weakened and paralyzed muscles were used in a number of cases.

Of all available methods of physical therapy, hydrotherapy is the safest and offers greatest benefit. Its advantages are:

1. It is more restful than dry heat.
2. It permits more active exercise with less fatigue.
3. In active motion of the parts the child automatically refrains from overstretching the muscles.

Exercises out of water are begun when the patient can spend one-half hour in the tub without fatigue. Even then the exercises are limited to one or two motions of the part. These exercises are gradually increased as muscular strength improves. SCHLUTZ, Chicago.

TREATMENT OF MENINGOCOCCIC MENINGITIS. E. S. BREWSTER, West Virginia M. J. **35**:224 (May) 1939.

Four weapons are available for treatment of meningococcic meningitis: lumbar puncture, antiserum, antitoxin and sulfanilamide. (The author would now doubtless add sulfapyridine.)

A boy aged 4 months with meningococcic meningitis is described. The diagnosis was established by laboratory methods.

Four thousand units of specific antitoxin was given into the right external jugular vein and 2,000 units was given intravenously by way of the superior longitudinal sinus. Spinal drainage was done fifteen times. Sulfanilamide to a total of 355 grains (22.9 Gm.) was given. The average daily dose was 20 grains (1.29 Gm.). Supportive treatment was given in the form of iron and of vitamins A, C and D.

Recovery was complete in twenty days. SHAWKEY, Charleston, W. Va.

CONVALESCENT SERUM IN CONTAGIOUS DISEASES. JAMES A. CONNER, Wisconsin M. J. **38**:638 (Aug.) 1939.

A review of the indications for and advantages of the use of convalescent serum in prevention and treatment of contagious diseases is given. Blood should be taken from the donor within six months after his illness.

Scarlet fever convalescent serum is believed to protect about 96 per cent of susceptible persons. It is of value also in active therapy.

Modification of measles by the use of measles convalescent serum is discussed as well as the more questionable value of convalescent serums in the modification and prevention of other diseases. JANNEY, Wauwatosa, Wis.

SEROLOGIC TYPES OF STREPTOCOCCI ASSOCIATED WITH SCARLET FEVER IN ADELAIDE. E. V. KOEGH, I. MACDONALD, J. BATTLE and M. C. PUCKEY, M. J. Australia **1**:792 (May 27) 1939.

Of 78 streptococci isolated from patients with scarlet fever in Adelaide from September 1938 to February 1939, 55 were type "Wade," 17 belonged to other serologic types and 6 could not be typed. These findings from Adelaide in conjunction with those reported from Sydney and Melbourne show that distribution of the types of streptococci associated with scarlet fever varies in each of the three cities. The findings at Adelaide tend to confirm a previous impression that in a large city scarlet fever due to predominant epidemic strains is more severe than sporadic scarlet fever caused by endemic strains. GONCE, Madison, Wis.

CROSS INFECTION WITH CORYNEBACTERIUM DIPHTHERIAE IN DIPHTHERIA WARDS. HAROLD WILSON, M. J. Australia **1**:861 (June 10) 1939.

Studies were conducted in the Royal North Shore Hospital at Sydney, Australia, to determine the incidence of cross infection in wards for patients with diphtheria. It was found that where free mingling of patients was permitted cross infection with the three types—gravis, intermedius and mitis—was common, but where isolation could be maintained it was infrequent. The length of time required for sterile cultures and discharge from the hospital was greater in the nonisolated group, indicating that cross infection may be an important factor in the duration of hospitalization. TENNEY, Madison, Wis.

UNRECOGNIZED FORMS OF MEASLES ENCEPHALITIS. V. DE LAVERGNE and HELLUY, Progrès méd., March 11, 1939, p. 337.

The authors first discuss circumstances which render recognition of encephalitis associated with measles difficult. For example, it may occur with other complications such as pneumonia, and the somnolence and convulsions may be interpreted as part of that disease. It may show itself as ataxia or a nocturnal delirium which lasts only for a few nights. Occasionally it is late in manifesting itself, and any direct connection with the measles may be difficult to establish. For these reasons careful examination of the nervous system is advisable.

The principal pathologic characteristics are said to be vasodilatation with hemorrhages and wide dissemination of the lesions. However, there may be an extensive hemorrhage in one area, or the lesions may be confined mostly to one area, such as the cerebellum. Blindness and epilepsy are cited as possible permanent sequelae.

The authors think that encephalitis is a more common complication of measles than has been suspected. LESLIE, Evanston, Ill.

ENCEPHALOMYELITIS FOLLOWING VACCINATION. A. PICCIOLI, Lattante **10**:231 (June) 1939.

A boy aged 4 years was vaccinated on May 25. The vaccination ran a normal course. The boy had mild fever. On the morning of June 5, high fever, pain in the arm and restlessness appeared, followed by paralysis of the left arm and left leg. Power returned to the left leg, but the left arm and shoulder were still paralyzed sixteen months later. There had been a few cases of poliomyelitis in the community at the time of the illness. In retrospect, it is hard to differentiate between polioencephalomyelitis and vaccinal encephalomyelitis. Piccioli suggests that vaccination be postponed when acute poliomyelitis is epidemic.

HIGGINS, Boston.

ACUTE ANTERIOR POLIOMYELITIS: ITS PRESENT CLINICAL STATUS. G. DE TONI, Lattante **10**:319 (Aug.) 1939.

De Toni reviews the recent literature on poliomyelitis and outlines the different types of that disease. HIGGINS, Boston.

ETIOPATHOGENETIC, EPIDEMIOLOGIC AND PROPHYLACTIC PROBLEMS OF ACUTE ANTERIOR POLIOMYELITIS. A. MONTAGNA, Lattante **10**:331 (Aug.) 1939.

The yearly average number of cases of poliomyelitis in Italy has increased from 644 in the period from 1924 to 1935 to 2,340 in that from 1936 to 1938. In the period from 1933 to 1935 it was 750; the rate was 18 per million inhabitants, considerably lower than in other countries (Denmark, 495; Sweden, 142; Norway, 82; Finland, 72; United States, 61).

An extensive study is being made of epidemics now in progress (1939) in the provinces of Arezzo, Bologna, Mantova and Modena.

The recent literature on poliomyelitis is reviewed.

HIGGINS, Boston.

CLINICAL STATISTICS ON POLIOMYELITIS. R. CANNATA, Med. inf., Rome **10**:225 (Aug.) 1939.

Statistics are given regarding the cases of infantile paralysis seen at the pediatric clinic and infants' hospital in Palermo, Sicily. Two hundred and four cases were observed in six years. It is surprising that of the seventy-two months reviewed there were only eight in which there were no cases of infantile paralysis. The distribution according to seasons was: winter, 23; spring, 59; summer, 72, and fall, 50. The maximum number of cases in any one year was 75 and the minimum 18. Practically all of the patients had paralysis, those with abortive forms not being included in the list. Only 9 of the patients died (4.5 per cent). Twenty-six showed complete cure of the paralysis. Three died of complicating bronchopneumonia or diphtheria.

HIGGINS, Boston.

INTUBATION IN THE TREATMENT OF DIPHTHERITIC CROUP IN CHILDREN. G. DI GIOVINE, Med. inf., Rome **10**:232 (Aug.) 1939.

Di Giovine reviews intubation, its technic, its probable complications and its contraindications in the treatment of patients with diphtheritic stenosis of the larynx. He concludes that for the most part intubation is preferable to tracheotomy.

HIGGINS, Boston.

INFANTILE PARALYSIS, A SOCIAL DISEASE. SERGIO PICCINI, Med. ital. **20**:70 (Feb. 28) 1939.

The social character of a disease is determined by the possibility of preventing it and combating it with legislative means and also by its extent and harmfulness. From an extensive bibliography the author cites the reports which he considers most important. He reports 64 cases of infantile paralysis and classifies the condition into eight forms. The most common is the spinal form. There are, in addition, an ascending type of Landry's paralysis, a bulbopontile form, an encephalic form, an ataxic form, a neuritis form, a meningeal form and an abortive form. Before nervous or paralytic signs appear, a gastrointestinal form, an anginal form, an influenzal form, a painful form, a sweating form, an exenthematic form, a respiratory form and a pseudocroupous form of the disease may be recognized.

The epidemiology is discussed. A table on mortality and morbidity is added. Ninety-three per cent of the author's patients were in the first four years of life, the greater number being in the first two years. Sixty-one per cent were boys and 39 per cent girls.

Under the heading of prophylaxis the author stresses isolation of the patient, terminal disinfection, boiling of all water and milk, a good feeding hygiene, convalescent serum, "parents' whole blood vitaminization" and mild nasal antiseptics. The population must be educated to cooperate with the sanitary authorities.

MARTOCCIO, Utica, N. Y.

INFANTILE PARALYSIS, A SOCIAL DISEASE. SERGIO PICCINI, Med. ital. **20**:118 (March 31) 1939.

This is a continuation of a previous article (*Med. ital.* **20**:70, 1939). The author takes up the subject of therapy and discusses the various forms which have been tried. Serum therapy, vaccine therapy, chemotherapy and heat therapy have all been used. The author concludes that symptomatic therapy is the form to be preferred at present. The pathologic physiology is discussed briefly. Prevention of deformities is the main essential in therapy. Orthopedic treatment of deformities and physical therapy are considered. The latter should be used for at least two years.

The author discusses the part that Italy is playing in combating the disease and assisting those afflicted with it.

MARTOCCIO, Utica, N. Y.

RESEARCHES ON THE TIME OF APPEARANCE OF BLANCHING ABILITY OF THE SERUM OF PATIENTS WITH SCARLET FEVER. F. TECILAZIC and P. FRANZINI, Med. ital. **20**:186 (April 30) 1939.

The authors report their investigations of the time of appearance of blanching ability of the serum of patients with scarlet fever. Twenty-nine patients with mild scarlet fever were studied. The technic of the Schultz-Charlton test was used. The authors conclude that the serum can acquire this ability in seven to eight days. In more than half of the cases this occurred before the fourteenth day. There is no relation between the intensity of the disease and the time of appearance of blanching ability.

MARTOCCIO, Utica, N. Y.

MALARIAL THERAPY IN POLIOMYELITIS EVALUATED FROM OBSERVATION OF SEVERAL CASES OF ASSOCIATED MALARIA AND ACUTE POLIOMYELITIS. A. CHIEFFI, Riv. di clin. pediat. **37**:289 (April) 1939.

Recently Garofali has claimed that malarial attacks counteract the acute phases of poliomyelitis, and Kanders has maintained that inoculation with malaria is beneficial to paralyzed patients thirty to sixty days after the acute stage.

Chieffi reports 6 cases of associated malaria and poliomyelitis. No benefit due to malaria could be noticed either in the acute or in the paralytic stage of the poliomyelitis. One child's death (from Landry's paralysis) was certainly hastened by the malarial paroxysms.

HIGGINS, Boston.

BEHAVIOR OF NONPROTEIN NITROGEN IN THE BLOOD DURING DIPHTHERIA AND SCARLET FEVER. L. NASSI, Riv. di clin. pediat. **37**:300 (April) 1939.

Nassi presents his own technic for determining the level of nonprotein nitrogen in the blood.

From observations on 14 patients with diphtheria he concludes that the nonprotein nitrogen content of the blood increases with the malignancy of the disease and with the amount of damage to the tissues by toxin.

From observations on 16 cases of scarlet fever without nephritis he found there was a rise in the nonprotein nitrogen content of the blood in the third to the fifth week of the disease. This may indicate latent renal disease. Vitamin C should be of therapeutic benefit in such cases, as well as in cases of definite nephritis, in maintaining the capillaries intact.

HIGGINS, Boston.

ROENTGEN TREATMENT OF DIPHTHERIA CARRIERS. E. MESSERI, Riv. di clin. pediat. **37**:337 (April) 1939.

Messeri claims 100 per cent success for roentgen treatment in ridding carriers of diphtheria bacilli. Twenty-two cases are reported. The technic is described in detail.

HIGGINS, Boston.

GLUTATHIONE IN THE BLOOD DURING MEASLES. E. MESSERI, Riv. di clin. pediat. **37**:613 (July) 1939.

Observations on 20 children with measles revealed that the glutathione content of the blood was definitely increased by from 20 to 90 per cent during the eruptive stage. The increase was proportional to the intensity of the rash.

HIGGINS, Boston.

SCARLET FEVER AGAIN BECOMES MORE SEVERE. H. G. HUBER, Kinderärztl. Praxis **10**:297 (July) 1939.

For the last two years the author has observed more frequent and more severe cases of scarlet fever in Chemnitz, Germany. He sees an acute increase in the incidence of the toxic and of the septic form. HEYMANN, Cleveland.

Acute Infectious Diseases

THE INCIDENCE OF INTESTINAL PARASITES. AN ANALYSIS OF 2,265 ROUTINE, CONSECUTIVE STOOL EXAMINATIONS IN THE OUTPATIENT DISPENSARIES OF CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS. EMMA S. MOSS, Am. J. Clin. Path. **9**:437 (July) 1939.

The author includes in this study figures on 146 cases of infestation with Ascaris. In 138 cases the condition occurred in patients under 16 years of age. The sex and race incidence were practically parallel in this group and in the older group, but the overwhelming predilection of the condition for the younger group is clearly shown. WEIGAND, Indianapolis.

MANAGEMENT OF CHILDREN WITH SUBACUTE RHEUMATIC FEVER. VALENTINE P. WASSON, Arch. Pediat. **56**:553 (Sept.) 1939.

The most important single factor in the regimen of a rheumatic child is mental and physical rest, which, unfortunately, in many instances is difficult to obtain because of economic and moral uncertainty, overcrowding and lack of parental discipline.

A good diet is most essential, and with patients of good economic and social status this is usually no problem. However, for the very poor, proper diet frequently is a distressing problem. This may be vastly improved through the efforts of the social agencies in supplying extra milk, iron and cod liver oil.

The problem of physical exercise is of paramount importance. Physicians who adopt an ultraconservative course in prescribing little or no exercise do considerable harm to the patient's heart, body and mind. Hard and fast rules cannot be laid down, for the amount of exercise is not dependent on the number of murmurs or the type of murmurs but on the condition of the myocardium and on the possibility of an infection that may have a possible relation to a recrudescence of rheumatic fever.

A patient recovering from rheumatic fever should be barred from all exercise until absence of an active process is fully established and should not be allowed out of bed until the temperature, leukocyte count, Schilling count and sedimentation rate are within normal limits. From then on gradual training of the heart muscle should begin. A well trained patient with cardiac disease learns to exercise to the limit of his tolerance.

The object of a clinic for patients with rheumatic heart disease should be rehabilitation of the patient with an injured heart and protection of the heart against further infections. A close, systematic watch should be kept on each patient. A physician who is satisfied with listening to murmurs and who sees the patient once in three or six months misses many important symptoms in an insidious disease like rheumatic fever. ORR, Buffalo.

FURTHER OBSERVATIONS ON THE OCCURRENCE OF RHEUMATIC MANIFESTATIONS IN THE FAMILIES OF RHEUMATIC PATIENTS. ROSS L. GAULD, ANTONIO CIOCCO and FRANCES E. M. READ, J. Clin. Investigation **18**:213 (March) 1939.

In the families of 96 rheumatic children, the percentage of persons with a rheumatic history was found to be consistent in two generations and was 3.7 times as high as was found in a group of control families. When one or both parents had a history of rheumatic manifestations, a greater percentage of the offspring was rheumatic than of the offspring of parents who gave no history of rheumatic disease. Among the offspring of rheumatic mothers, the percentage of girls with rheumatic manifestations was almost twice as high as the percentage of boys so affected. A greater percentage of persons with rheumatic disease was found among the maternal aunts and uncles than among the paternal aunts and uncles of the rheumatic children. These findings suggest that hereditary constitution may play a role in the predisposition to this disease.

GUEST, Cincinnati.

PRACTICAL EPIDEMIOLOGY OF GONOCOCCIC INFECTIONS IN CHILDREN. THEODORE ROSENTHAL and JOSEPH WEINSTEIN, New York State J. Med. **39**:718 (April 1) 1939.

Gonorrhea in children has long been recognized as an epidemic disease and a social problem. In New York city reported cases were investigated by either epidemiologists (some nurses and some physicians) or trained male investigators. It is found that the disease is most prevalent among dwellers of overcrowded tenements living under insanitary conditions. Although it occurs more frequently in little girls, it may occur in boys. Localized outbreaks can usually be traced to some adult.

AIKMAN, Rochester, N. Y.

DIAGNOSIS OF ECHINOCOCCAL (HYDATID) DISEASE IN MAN BY INTRADERMAL REACTION TO RABBIT CYSTICERCUS ANTIGEN. HARRY M. ROSE and JAMES T. CULBERTSON, Proc. Soc. Exper. Biol. & Med. **41**:426 (June) 1939.

Intradermal injection of 0.1 cc. to 0.5 cc. of phenolized fluid obtained from a fertile hydatid cyst of either sheep or man, because of the antigen present, produces either immediate or delayed cutaneous reaction with wheals and erythema in patients with suspected hydatid infections. An extract of Cysticerci of the common rabbit cestode Taenia pisiformis was used as an antigen and was found to produce positive reactions in 4 patients known to have hydatid disease. Ten normal men and 10 normal women serving as controls exhibited at most slight reactions and were considered "negative."

HANSEN, Minneapolis.

TYPHOID AND PARATYPHOID FEVER IN CHILDREN. MARCUS E. FARRELL, West Virginia M. J. **35**:227 (May) 1939.

Typhoid and paratyphoid fever are neither rare in infants nor uncommon in children. Paratyphoid A is rare, but paratyphoid B is not. Symptoms may be mild or absent, though the organisms may continue to be found in the stools for some time. Certain cases of "summer flu" and dysentery are really cases of paratyphoid. A correct diagnosis is important, especially from the standpoint of public health.

Early in typhoid fever the blood culture yields bacteria in 90 per cent of cases. The Widal test is less reliable and gives negative results for at least the first ten days. The Widal reaction may be positive because of a previous attack or because of inoculation.

Leukopenia may not develop until the second week. Relapses are uncommon but do occur. One should be conscious of typhoid and paratyphoid in the presence of obscure gastrointestinal infections in children.

Preventive inoculations are urged. More laboratory study is advised. The importance of remembering the tendency to relapse is stressed.

SHAWKEY, Charleston, W. Va.

REPORT ON EXAMINATION OF THE SPLEEN—AND PARASITE—RATES IN SCHOOL CHILDREN IN FREETOWN, SIERRA LEONE. H. PEASTON and E. A. RENNER, Ann. Trop. Med. **33**:49 (March 31) 1939.

The results of this investigation show that there has been a marked decrease in the incidence of malaria in the hyperendemic area of Freetown, as compared with MacDonald's figures (*Ann. Trop. Med.* **20**:239, 1926). Peaston and Renner examined 951 African children 3 to 10 years of age in nine Freetown schools, in November and December 1935 (early part of the dry season). They recorded the presence and size of enlarged spleens and the presence of malaria parasites in the peripheral blood (thick smears). As their records showed, about half of the children came from the western part of the town (hyperendemic area) and the other half from the central and eastern portion (endemic area). Of the whole series, 43 per cent had enlarged spleens (endemic 44.1 per cent, hyperendemic 42.6 per cent); 36.3 per cent showed parasites in the peripheral blood (endemic 34 per cent, hyperendemic 38.5 per cent). The true infection rate, which was calculated from the formula $p \frac{100}{x}$ (p = parasite-rate per cent, x = parasite-rate with enlarged spleen) was 80.8 (endemic 87.2, hyperendemic 75.9).

A brief account was given of the completion of the permanent canalization of the Sander's Brook main canal. The improved drainage scheme has done away with the hyperendemic area, so that now all Freetown should be regarded as an endemic area.

FREDEEN, Kansas City, Mo.

THE RELATIONSHIP BETWEEN ACUTE RHEUMATISM AND STREPTOCOCCAL ANTIFIBRINOLYSIN. C. BRUCE PERRY, Arch. Dis. Childhood **14**:32 (March) 1939.

From this investigation it is clear that the appearance of resistance to streptococcic fibrinolysin in the blood of patients with acute rheumatism is not an essential part of the rheumatic process. Antifibrinolysin is present in about 78 per cent of patients with acute rheumatism, which is approximately the proportion of normal patients who show antifibrinolysin after a streptococcic infection. There is no correlation between the duration of the presence of antifibrinolysin and the duration of the rheumatic attack or between antifibrinolysin and the severity of the rheumatic attack. There is some evidence that administration of salicylates after a streptococcic infection inhibits the formation of antifibrinolysin.

KELLY, Milwaukee.

SEARCH FOR IMMUNOLOGICAL RELATION BETWEEN "Q" FEVER AND OTHER RICKETTSIOSES. F. M. BURNET, M. FREEMAN, E. H. DERRICK and D. J. W. SMITH, M. J. Australia **2**:51 (July 8) 1939.

Serum from patients or animals suffering from most of the known rickettsioses were tested for agglutination with an emulsion of Rickettsia burneti, with negative results. Guinea pigs inoculated with a vaccine which will protect against Rocky Mountain spotted fever were not thereby protected against "Q" fever. Although the results show no evidence of immunologic relation between "Q" fever and the rickettsioses studied, attention is drawn to work by Dyer indicating a relation between "Q" fever and an infection obtained from ticks in Montana.

GONCE, Madison, Wis.

STREPTOCOCCIC BACTERIEMIA. HAROLD WATTS, Proc. Roy. Soc. Med. **32**:607 (April) 1939.

A boy aged 6 years was admitted to the hospital with a history of seven days' fever, sweating and earache. Three days before the onset of symptoms he had sustained a puncture wound of a finger, which healed by first intention. On examination some inflammation was found in both ear drums; the spleen was enlarged, and there was a soft systolic murmur at the apex. There was no clubbing of the fingers. Culture of the blood showed a pure growth of hemolytic streptococci in fifteen hours, but four days later a culture was sterile. The throat swab showed staphylococci and Micrococcus catarrhalis. Various blood counts and the treatment are given in the report. The author states that the most striking feature of the case was that in spite of a high swinging temperature the child never looked ill and did not complain of anything while in the hospital.

WILLIAMSON, New Orleans.

HUMAN BRUCELLOSIS AND ABORTION. M. JANBON and J. CADERAS DE KERLEAU, Presse méd. **47**:453 (March 25) 1939.

The authors point out that abortion as a result of brucellosis is rare in women. They present a case of a woman two months pregnant who aborted at about the fiftieth day of her disease, after three weeks of an afebrile phase. Brucella melitensis was recovered from the mother's blood, the blood of the fetal heart, the contents of the fetal stomach, the fetal liver and spleen, the placenta and the maternal uterine mucosa.

DAFFINEE, Boston.

BACILLARY DYSENTERY AS A CAUSE OF DEATH IN THE FIRST TWO YEARS OF LIFE. C. CAMMARELLA, Lattante **10**:248 (June) 1939.

This short paper reviews bacillary dysentery as a preventable cause of infant mortality. Forty-four per cent of the infants with dysentery observed by Cammarella have died. Prophylaxis offers the best attack on this mortality rate.

HIGGINS, Boston.

PSYCHONEUROTIC SYNDROME FROM MALARIA IN A CHILD. A. PAFUMI and C. GRILLO, Med. inf., Rome **10**:97 (April) 1939.

A 12 year old boy had had periods of chills and fever for three months which proved to be due to tertian malaria. During this period he showed interesting neurologic and psychiatric symptoms. The neurologic symptoms were convulsive episodes and ataxia during the febrile attacks. The psychiatric manifestations were those of depression. The boy wished to be alone and refused to eat for fear the food had been poisoned. These symptoms disappeared as the malaria responded to quinine therapy.

HIGGINS, Boston.

COMPARATIVE STUDIES OF PERIPHERAL BLOOD, MARROW BLOOD AND SPLENIC BLOOD IN VISCERAL LEISHMANIASIS. I. GATTO, Med. inf., Rome **10**:125 (May) 1939.

Blood from the spleen was obtained by splenic puncture. Blood from the bone marrow was obtained by puncture of the tibia. Observations were made on the blood of 10 children with kala-azar. The number of red cells, white cells and platelets in the splenic blood was in each case larger than in the peripheral or the marrow blood; thus the corpuscular elements of the blood seem to accumulate in the spleen in this disease. The red blood cells from the marrow showed less fragility than did those from the spleen or those from the peripheral capillaries; thus it appears that new red cells are less easily laked by hypotonic solutions.

However, the red cells obtained by splenic puncture also were less fragile than the cells of peripheral capillary blood; thus it would appear that the old red cells are not necessarily the most fragile.

HIGGINS, Boston.

SHIGA-KRAUSE DYSENTERY IN AN INFANT. U. MAROCCHETTI, Med. inf., Rome **10**:179 (June) 1939.

An infant aged 6 months was first believed to have simple summer diarrhea, but with persistent recurrences and passage of stools containing blood, mucus and pus, a diagnosis of bacillary dysentery was made. Dysentery bacilli of the Shiga-Krause type were obtained on culture of the stool. Further cases in the family brought out that the source of the illness was a boy with diarrhea who had recently returned from Africa. The author warns that serious diarrhea in infants may be traced to soldiers returning from Africa after having suffered dysentery there.

HIGGINS, Boston.

DYSENTERY (FLEXNER GROUP, TYPE Z) IN AN INFANT AGED ELEVEN MONTHS. J. SCHWARZENBERG L. and J. COUSINO A., Rev. chilena de pediat. **10**:154 (March) 1939.

The authors point out the difficulties of diagnosis of this condition in the very young infant and the great importance of systematic bacteriologic examination of the stools. This is often the only way in which the differential diagnosis between enterocolitis and enterocatarrh or gastrointestinal intoxication can be made.

SCHLUTZ, Chicago.

TAENIA INFESTATION: REPORT BASED ON EXAMINATIONS OF THE STOOLS OF 56,286 PATIENTS IN PEIPING UNION MEDICAL COLLEGE. L. S. WU, Chinese M. J. **55**:561 (June) 1939.

Children in infancy are rarely infested with Taenia because they receive little meat in the diet. The author reports the case of an infant 1 year of age. There were 21 children from 1 to 9 years of age in a total of 337 cases of infestations found by routine examination of the stools.

NEFF, Kansas City, Mo.

COINCIDENCE OF HUMAN KALA-AZAR AND CANINE LEISHMANIASIS WITH CUTANEOUS LESIONS IN A HOUSEHOLD. E. A. HO, Chinese M. J. **55**:566 (June) 1939.

The author reports 2 instances of canine infection, 1 being present for about one year before the appearance in the household of symptoms in a girl aged 2 years and 5 months. Other members of the family were not affected. The diagnosis was made by sternal puncture, which revealed Leishmania.

NEFF, Kansas City, Mo.

FURTHER STUDIES ON THE SERUM TREATMENT OF TYPHOID FEVER. H. YÜ, Chinese M. J. **56**:29 (July) 1939.

Concentrated antityphoid serum was prepared at the Henry Lester Institute of Medical Research, Shanghai, China, and used in the treatment of 64 patients with typhoid fever. The serum was given intramuscularly, the dose being 10 to 20 cc. for children and 40 cc. in adults. The treatment was begun at the earliest possible moment and repeated on three successive days. Thirty-three patients showed a prompt drop in fever and a decrease in toxic symptoms. The author feels that its value is substantiated and confirms the reports of Felix and other English workers.

NEFF, Kansas City, Mo.

Chronic Infectious Diseases

THE TUBERCULIN PATCH TEST: A COMPARISON WITH THE MANTOUX INTRACUTANEOUS TEST. G. TAYLOR, Am. Rev. Tuberc. **40**:236 (Aug.) 1939.

A comparison of the results of the patch test with those of the Mantoux test (0.1 mg. of old tuberculin) was made in 744 persons, ranging in age from 3 months to 71 years. The tests were comparable in 94.3 per cent of the entire group. Young adults were apparently more sensitive to the patch test than to the Mantoux test, while the opposite was true of persons above 50 years of age.

EUGENE H. SMITH, Ogden, Utah.

THE CUBAN NATIONAL TUBERCULOSIS CASE-FINDING CAMPAIGN: I. INTRODUCTION AND RESULTS OF TUBERCULIN TESTS. II. RESULTS OF X-RAY STUDIES. J. R. MENCIA, M. C. KAHN and E. MAYER, Am. Rev. Tuberc. **40**:522 (Nov.); 536 (Nov.) 1939.

I. The number of deaths from tuberculosis in Cuba is about 5,000 each year, and there are probably 40,000 persons with the disease in an advanced stage in the island. Only in comparatively recent years have effective efforts been made to control the disease. The government and private agencies are now actively cooperating in this work. As a part of this endeavor, a case-finding campaign was undertaken in a district of Habana comprising 150,000 persons of the middle and lower classes. To date 27,000 tests with purified protein derivative have been made, and over 10,000 roentgenograms. A second skin test was made on all whose tests were negative, except children under 5 years, and one half of the second dose was given to children between 5 and 10 years of age. Physical and laboratory examinations were made of those with positive reactions. Of the total number tested, 69.42 per cent gave positive reactions. The percentage was practically the same in whites, Negroes and mulattoes. Chinese males showed the highest percentage of reactors, and Chinese females, the lowest. The results in children were:

	Percentage with Positive Reactions
0 to 1 year	15.98
1 to 4 years	18.88
5 to 9 years	56.90
10 to 14 years	71.13
15 to 19 years	79.89

Among children under 1 year of age the white children gave the larger percentage of positive reactions; among those over 1 year, the percentage was slightly higher in Negroes.

II. In a case-finding survey, roentgen examinations (paper films) of 10,949 persons with positive reactions to tuberculin were made. The lesions found were classified as follows:

1. Residual. These were represented by calcified nodes, single or multiple; a primary complex associated with calcification of tracheobronchial or mediastinal nodes, fibrotic scars or pleurodiaphragmatic adhesions.

2. Latent. In this type there were potentially progressive shadows, latent infiltration of a first infection or childhood infiltration of a soft flocculent and strandlike nature or latent apical reinfection or an adult type of tuberculosis.

3. Manifest. This type consisted of massive caseous tuberculosis, of the adult type and reinfection type, with or without sputum containing the bacilli.

Of the 10,949 persons examined, 10.38 per cent showed roentgen evidence of disease, classified as follows: residual, 41.38 per cent; latent, 32.48 per cent; manifest, 26.14 per cent. With the patients grouped according to age, tuberculous lesions were found as follows: at ages of 20 to 34 years, in 50 per cent; at 10 to 14

years, in 25 per cent; at 5 to 9 years, in 13.3 per cent; at 15 to 19 years, in 7.83 per cent. A slightly higher percentage of the females showed pulmonary disease.

EUGENE H. SMITH, Ogden, Utah.

THE TUBERCULIN TEST: ITS VALUE AND ITS LIMITATIONS. E. R. LONG, Am. Rev. Tuberc. **40**:607 (Dec.) 1939.

The test referred to in the title is the intracutaneous test using old tuberculin purified protein derivative. It was sought to determine the value of the procedure in mass case finding. "Case" in this connection was employed to designate only a case in which tuberculous infection had progressed to a point at which symptoms or roentgen evidence of disease had developed.

Compared with roentgen findings, intracutaneous tests with reliable material will indicate the presence of infection in 90 to 95 per cent of cases in ordinary practice. Two principal sources of error exist if reliance is placed on skin testing alone. In certain subjects the development of cutaneous sensitivity is delayed, and tests made early in the course of the infection will be negative. Another group may show waning or disappearance of previous positive reactions. The errors which may result from loss of previous sensitivity are important and will probably increase with decline in the incidence of tuberculous infection. Unless allergy is stimulated from time to time by reinfection, the intensity of the response will diminish. From a practical standpoint, however, while the allergic response in 11 per cent of 2,490 cases changed from positive to negative in the course of ten years, in no instance did it become negative in the frank reinfection type of pulmonary tuberculosis.

EUGENE H. SMITH, Ogden, Utah.

X-RAY FINDINGS IN TUBERCULIN REACTORS AND NONREACTORS. B. H. DOUGLAS, Am. Rev. Tuberc. **40**:621 (Dec.) 1939.

Since the object of mass surveys with tuberculin surveys is the finding of the active lesion, Douglas holds that, in spite of its limitations, skin testing remains the best method of screening the few from the many for more intensive study. The expense of the roentgen examination and the experience and ability required in the interpretation of the findings definitely limit the use of this type of investigation as at present developed. Of the tuberculin test it may be said that it is rarely negative in the presence of significant tuberculous infection.

EUGENE H. SMITH, Ogden, Utah.

VALUE OF TUBERCULIN SKIN TESTS IN A SCHOOL PROGRAM. W. E. AYLING, New York State J. Med. **39**:1463 (Aug. 1) 1939.

In Syracuse, N. Y., tuberculin tests made with 0.1 mg. of old tuberculin by the Mantoux method have been used in the high school for the past five years in finding cases of tuberculosis. The procedure has been to acquaint the pupils and parents with the importance of tuberculosis and with the tuberculin test. By disseminating information through the radio, the press, motion pictures, lectures and pamphlets, every effort was made to secure the cooperation of the school principal and the teaching staff. The students were then given a form letter about the test with a request slip attached. Fifty-four per cent of the parents registered gave permission for the examination and 10,392 tests were made. Of these, 32.9 per cent were positive; 90.6 per cent of the students who gave positive reactions were examined by roentgenograms; of the roentgenograms, 40.9 per cent were essentially negative; 50.9 per cent showed apparently healed childhood type of tuberculosis; 7.2 per cent were suggestive of activity and 0.9 per cent indicated that there was probably active disease. The students found to have tuberculosis were all referred back to the family physician, who had to examine other members of the household. The death rate from tuberculosis in Syracuse has dropped from 59 per hundred thousand in 1927 to 29 in 1937.

AIKMAN, Rochester, N. Y.

THE TREATMENT OF CONGENITAL SYPHILIS WITH AN INTRAVENOUS ARSENICAL: AN ANALYSIS OF TWO HUNDRED AND FOUR CLINICAL CASES. J. K. HOWLES, South. M. J. **32**:940 (Sept.) 1939.

The good results which follow the intravenous injection of mapharsen in the treatment of prenatal and congenital syphilis seem to merit the acceptance of that method as a standard mode of therapy in such cases. The addition of bismuth subsalicylate therapy to the mapharsen therapy produces even more satisfactory results.

The mildness of the local reactions which follow technical accidents with this mode of therapy furnishes an additional argument for its use as an intravenous arsenical agent.

The low incidence of severe reactions, whether immediate or delayed, is evidence of its high therapeutic value.

The combined method of treatment (mapharsen and bismuth subsalicylate) is further justified by the excellent tolerance to it on the part of children, whose visceral state is good.

The high incidence in Howles's series, of reversal in the Wassermann reaction of the blood can be attributed to the combined method of treatment. The excellent follow-up which he obtained also played an important part in his results.

SCHLUTZ, Chicago.

IS THE ROENTGENOGRAPHIC EXAMINATION OF THE LONG BONES MORE IMPORTANT FOR THE DIAGNOSIS OF CONGENITAL SYPHILIS THAN THE SEROLOGIC STUDY OF THE BLOOD? W. REINHARDT, Kinderärztl. Praxis **10**:197 (May) 1939.

Reinhardt answers his question in the negative. He observed infants in whom there was no evidence of syphilitic osteochondritis, although tests of the blood gave reactions positive for syphilis. He cites 3 cases. The patients were aged 9 days, 14 days and 2 months.

HEYMANN, Cleveland.

CHILDREN IN A HEALTH RESORT AND TUBERCULOSIS. J. W. SCHMITZ and J. BOS, Nederl. tijdschr. v. geneesk. **83**:2456 (May 27) 1939.

Results of research among children sent to a country health resort are reported. Tuberculin tests and roentgen examinations of these children showed that about 0.5 per cent were suffering from tuberculosis in an active form. This proves the necessity of roentgen examination of children before they are admitted to health resorts. Most of these children were not known before to suffer from tuberculosis. They were handed over to tuberculosis dispensaries, which made possible the tracing of these infections to their sources.

VAN CREVELD, Amsterdam, Netherlands.

DOES THE BOVINE TYPE OF THE TUBERCLE BACILLUS IN MAN GIVE RISE TO A DEFINITE FORM OF TUBERCULOSIS? G. J. HUËT, Nederl. tijdschr. v. geneesk. **83**:2744 (June 10) 1939.

Huët endeavored to answer this question by a study of 19 cases of bovine tuberculosis in man. He concludes: It is not probable that the bovine tubercle bacillus as such is less virulent than the human type. If bovine tuberculosis in general seems to follow a more benign course, this must be due to a difference in the routes of infection. Human bacilli inevitably invade the most vulnerable organ, the lung. In bovine infection, which invades the organism by a different route, there is a good chance that the organs infected will be those which have a better prognosis for overcoming the infection.

It is still unproved that tuberculosis caused by the bovine bacillus has a special tendency to be metastatic. Figures in favor of this assumption can be explained by the selection of the material from which they are derived.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Blood, Heart and Blood Vessels and Spleen

PRESENT KNOWLEDGE OF THE SOURCES AND ACTION OF COPPER IN NUTRITIONAL ANEMIA. C. P. SEGARD, Am. J. Digest. Dis. **6**:315 (July) 1939.

Evidence is submitted from a number of reliable sources that copper is necessary and a specific catalytic agent in the formation of hemoglobin. One cannot rely on the amount of copper present as a contaminant of therapeutic iron. Although copper is present in some foods in wider variation than iron, through processing this source of copper has been almost completely eliminated.

In dealing with nutritional anemia it is important to be certain of the presence of copper.

MORRISON, Savannah, Ga.

ROENTGENOLOGY OF CONGENITAL CARDIOVASCULAR DISEASE. H. ROESLER, Am. J. Roentgenol. **42**:72 (July) 1939.

Four groups of cardiovascular structural anomalies are discussed from a roentgenologic aspect:

1. Conditions without any roentgenologic evidence of an anomaly, such as a small interventricular septal defect.

2. Conditions characterized by deviations from the normal which are not characteristic for a congenital anomaly; for example, a three-chambered heart, which produces general cardiac enlargement, but so do many acquired diseases.

3. Conditions which reveal fairly characteristic features, such as: (1) the wooden shoe heart, with which anatomically one finds stenosis of the pulmonary artery associated with marked hypertrophy of the right ventricle, an interventricular septal defect and a dextroposition of the aortic orifice; (2) the long globular heart, with which the pulmonary artery and its branches reveal on roentgen examination exaggerated pulsations and marked enlargement of the pulmonary conus, and with which anatomically there is an interatrial septal defect, associated with dilatation both of the cavities on the right side of the heart and of the pulmonary artery as well as with hypoplasia of the aorta; (3) a condition which presents a cardiac shadow to the left and dorsad, indicating left ventricular preponderance, and in which the anatomic characteristic is atresia of the tricuspid orifice with defective development of the right ventricle.

4. Conditions with unequivocal roentgenologic features: (1) dextrocardia, (2) coarctation of the aorta and (3) a persistent right-sided aortic arch which crosses over the right bronchus and recrosses to the left, displacing the esophagus and trachea.

SQUIRE, Chicago.

MENTAL RETARDATION ASSOCIATED WITH CONGENITAL HEART DISEASE: A STUDY OF TWENTY-TWO CASES. M. ROSS, J. Pediat. **14**:21 (Jan.) 1939.

Twenty-two patients with congenital cardiac disease, ranging in age from 5 to 14 years, were given the Binet-Simon intelligence test. Two patients had intelligence quotients below 50, thus belonging to the imbecile group; 7 patients were in the moron group, with quotients between 51 and 70, and 8 were in the dull normal group with quotients between 71 and 90. Only 4 were in the normal range, that is, had intelligence quotients between 91 and 110, and the highest quotient in the group was 117. There was no relation between the type of cardiac lesion and the level of intelligence. This group of patients was compared with a cross section of patients from the Harriet Lane psychiatric dispensary. The chief difference was that among the patients with congenital cardiac disease a higher percentage fell into the moron group and a smaller percentage into the normal group than among the patients from the psychiatric dispensary.

RAUH, Cincinnati.

MICROSEDIMETER FOR ERYTHROCYTE SEDIMENTATION TEST. H. VOLLMER, New York State J. Med. **39**:1583 (Aug. 15) 1939.

A simple micromethod for determining the sedimentation rate of erythrocytes is described. The apparatus is small, and the procedure causes little pain or fear. It was first described by Langer and Schmidt. Vollmer has found it highly satisfactory.

AIKMAN, Rochester, N. Y.

THE PROTHROMBIN CONVERSION RATE IN VARIOUS SPECIES. E. D. WARNER, K. M. BRINKHOUS and H. P. SMITH, Proc. Soc. Exper. Biol. & Med. **40**:197 (Feb.) 1939.

The rate at which prothrombin is converted into thrombin is much slower in the plasmas of man and the guinea pig than in the plasmas of the dog, rabbit, cat, rat, fowl, turtle and fish. This slow conversion of prothrombin in man is undoubtedly a factor which aggravates hemorrhagic diseases.

HANSEN, Minneapolis.

DEVELOPMENT OF CARDIO-PERICARDIAL ADHESIONS FOLLOWING THE USE OF TALC. S. A. THOMPSON, Proc. Soc. Exper. Biol. & Med. **40**:260 (Feb.) 1939.

Hydrous magnesium silicate (talc) powder was placed in the pericardium in a series of 46 experiments on 40 dogs, with the production of adhesions of the pericardium and the epicardium. Adhesions occurred as early as after eighteen hours; by one week the surfaces were firmly adherent, and by four weeks the layers fused as one layer of tissue.

HANSEN, Minneapolis.

CONGENITAL LESIONS OF THE HEART. J. ODINET, Union méd. du Canada **68**: 614 (June) 1939.

This is a well written article summarizing present knowledge of congenital cardiac lesions of the most common types.

DAFFINEE, Boston.

CONGENITAL HEART DISEASE IN GENERAL PRACTICE. P. T. O'FARRELL, Irish J. M. Sc., September 1939, p. 645.

O'Farrell states that there are many types of congenital anomaly of the heart but that only a few can be diagnosed clinically. He states that bacterial endocarditis is a common secondary condition. He gives a classification of congenital heart disease and demonstrates certain lesions.

C. E. SNELLING, Toronto, Canada.

LEUKOCYTES OF THE BLOOD IN CASES OF SEVERE ANEMIA IN INFANTS. P. NOBÉCOURT, Arch. de méd. d. enf. **42**:481 (Aug.) 1939.

There is lack of agreement on the number of leukocytes and on the hemoleukocyte formula for the first two years of life. Extremely variable estimations are also given in discussions of the anemia occurring in that period. Nobécourt has made an exhaustive study of 23 cases of anemia in each of which the red cells numbered less than 3,000,000 per millimeter of blood.

His conclusions follow:

1. In observing cases of moderate anemia with a count of 2,000,000 to 3,000,000 red blood corpuscles, it is necessary to distinguish two groups of anemias: (1) the group in which the leukocytes appear in normal or nearly normal numbers, with the medullary series predominating over the lymphatic, but with no abnormal leukocytes, and (2) the group in which there are changes in the leukocyte count, which may be above or below the average, but with a constant predominance of the lymphatic types over the medullary.

2. In cases of grave anemia with a red blood cell count below 2,000,000 per millimeter, the number of white cells is never normal; it may vary from 3,000 to 66,560.

AMESSE, Denver.

BLOOD TRANSFUSION IN PEDIATRICS. J. BONABA and R. CHARIONE, Bol. Inst. internac. am. de protec. a la infancia **13**:244 (Oct.) 1939.

This is a discussion of the practicability of blood transfusions for infants and children.

LESLIE, Evanston, Ill.

VALUE OF CLINICAL AND ROENTGENOLOGIC DATA IN INFANTILE CARDIOPATHIES. B. DELGADO CORREA, Bol. Inst. internat. am. de protec. a la infancia **13**:267 (Oct.) 1939.

Infantile acquired cardiopathies are one of the most serious problems of the pediatric clinic owing to their increasing incidence, the serious injuries due to them, the invalidism that they cause in a great percentage of the affected children, and the high death rate, especially from those of rheumatic origin.

Of great importance is early diagnosis followed by adequate treatment in specialized clinics and polyclinics.

The congenital cardiac abnormalities are an important and intricate chapter of infantile pathology. Clinical and roentgen examinations make it possible to attain an exact diagnosis.

In clear cases of cardiac abnormality roentgen evidence of the alterations is almost constantly present.

In the continued study of patients there is evident agreement between the clinical and the roentgen findings. Improvements and aggravations of the conditions are shown in the roentgenograms. In many cases roentgen examination might determine details and incline diagnosis toward a certain disease.

FROM AUTHOR'S SUMMARY.

EXPERIMENTAL STUDIES ON THE POTASSIUM-CALCIUM QUOTIENT IN SERUM AND ON THE POTASSIUM-CALCIUM CONTENT OF THE GROWING FETUS AND THE INFLUENCE OF CALCIUM LACTATE AND ASCORBIC ACID ON THESE TWO FACTORS. H. WINKLER and H. FRITSCHE, Ztschr. f. Geburtsh. u. Gynäk. **118**: 405, 1939.

Since recent studies have shown the influence of vitamin C on the formation and the deposit of calcium in fractures, Winkler and Fritsche have investigated the relation of vitamin C to the calcium content of the growing fetus. They find that the potassium content of the serum and embryonic tissue decreases as pregnancy progresses.

Doses of calcium lactate and ascorbic acid have no influence on the potassium-calcium content of serum or tissue. There is no activiation of calcium deposits in the fetal skeleton after additional doses of vitamin C, nor is the potassium-calcium metabolism influenced by a diet free of vitamin C and calcium. The authors conclude that it is not possible to influence intrauterine growth by additional doses of calcium and vitamin C.

ADAIR and POTTER, Chicago.

HEMOPHILIA IN A GIRL. M. KUBO, Acta pædiat. japon. **45**:1116 (Aug.) 1939.

The patient was 6 years old. The diagnosis was made by elimination of other related conditions. There was a history of a familial tendency to bleed. The patient was referred to the author because of uncontrollable hemorrhage following a minor surgical treatment on the swollen and painful gum of the right lower jaw. The blood revealed marked anemia. The bleeding time was slightly prolonged (five to nine minutes), and there was a tendency for the platelet counts to be high (256, 300 to 525, 900). The coagulation time was definitely prolonged (incomplete clot formation in twenty-five to forty-five minutes and complete coagulation in one and a half to two hours). The clot retraction test, the value for fibrinogen (125 units per cubic centimeter) and that for serum calcium (9.4 mg. per hundred cubic centimeter) were normal. The Rumpel-Leede phenomenon

was negative. The results of these examinations ruled out congenital fibrinopenia, thrombopenia, hereditary thrombopathy, phosphorus poisoning and cholemia, leaving hemophilia as the only possibility. KATO, Chicago.

ACCIDENTS PRODUCED BY BLOOD TRANSFUSIONS. D. TIMUS, Med. in., Bucuresti **1**:180 (March-April) 1939.

Since transfusion of blood is a procedure very frequently employed in the treatment of adults and children, Timus calls attention to the accidents which may occur, citing 2 cases. In the first case tertian malaria was transmitted to an infant of 5½ months who was suffering from toxic bronchopneumonia. In the second case syphilis was transmitted by transfusion to a woman who presented postpartum hemorrhage. Timus concludes that when it is necessary to do a transfusion, however urgent it may be, it is necessary not to be content with determining the blood group but to have at the same time rigorous control of the donor from the somatic and serologic standpoint. FROM THE AUTHOR'S SUMMARY.

STENOSIS OF THE ISTHMUS AORTAE. P. H. KRAMER, Nederl. tijdschr. v. geneesk. **83**:4208 (Aug. 26) 1939.

This is a clinical and pathologic report on a man who died at the age of 30 years from coarctation of the aorta and open ventricular septum. Moreover, at the postmortem examination a patent ductus Botalli and biscuspid aortic ostium were found. The cause of death was septic endocarditis.

VAN CREVELD, Amsterdam, Netherlands.

DETERMINATION OF BLOOD VOLUME BY MEANS OF A BLUE DYE. E. LOPES CARDOZO, Nederl. tijdschr. v. geneesk. **83**:5357 (Nov. 11) 1939.

The blue dye T 1824 has distinct advantage over congo red for determining plasma volume. The method is described in detail, and stress is laid on a reliable estimation of the concentration of the dye in the serum. For this purpose the use of a good photoelectric cell is advised. Some characteristic changes of plasma volume in various diseases are illustrated.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of Nose, Throat and Ear

NASOPHARYNGEAL ATRESIA: DESCRIPTION OF AN OPERATION. HENRY M. GOODYEAR, Arch. Otolaryng. **29**:974 (June) 1939.

The author notes the infrequent occurrence of nasopharyngeal atresia but stresses its serious complications and presents a simple operative procedure which produces excellent results if care is taken to preserve sufficient mucous membrane.

GREENWOOD, Chicago.

CONGENITAL PREAURICULAR SINUS. REED DINGMAN, Arch. Otolaryng. **29**:982 (June) 1939.

The clinical signs of preauricular sinus are presented, and the possibility of its resulting from a disturbance in the closure of the first branchial cleft is discussed. Surgical excision preceded by several sinal irrigations is the advised course of treatment. Attention is called to the importance of removing the original sinus and its tract in addition to the draining fistula to insure complete recovery. GREENWOOD, Chicago.

CONGENITAL BRANCHIOGENIC PREAURICULAR SINUS: A NOTE REGARDING ITS TREATMENT. FRED Z. HAVENS, Arch. Otolaryng. **29**:985 (June) 1939.

Contributing factors in the development of a branchiogenic preauricular sinus are discussed. Attention is called to the locality as a frequent host to infection. A procedure which has been successfully applied in cases of repeatedly infected preauricular sinuses is outlined.

GREENWOOD, Chicago.

OBSCURE LOW GRADE CHRONIC INFECTIONS OF THE ETHMOID SINUSES. GEORGE E. SHAMBAUGH JR., J. A. M. A. **112**:1226 (April 1) 1939.

Shambaugh describes five types of obscure chronic infections of the ethmoid sinuses according to the presenting symptoms. The diagnosis is confirmed by the findings after Proetz displacement suction has demonstrated the presence of pus in the sinus. Treatment consists of removal of the accumulated material by the same method.

Of the five types of symptoms presented, i. e., headache, focal infection, chronic cough, frequent head colds and unexplained fever, two were seen in children. Persistent cough and unexplained fever were shown to be due in some instances to this condition and were cured by repeated drainage of the ethmoid sinuses.

HEERSMA, Kalamazoo, Mich.

THE PRESENT POSITION OF TONSIL OPERATIONS. MACLEOD YEARSLEY, Brit. J. Child. Dis. **36**:1 (Jan.-March) 1939.

"It would appear that large numbers of healthy tonsils are sacrificed annually, by over-zealous operation, in a slaughter of the innocents which out-Herods Herod."

A good part of the article consists of quotations from Alison Glover's paper on "The Incidence of Tonsillectomy in School Children." "If, as some believe, the tonsils have a protective function . . . the child's entry to the environment of school might seem the time when they are most likely to be useful."

To remove tonsils is often a blunder, the real trouble being in the nose. The two points of importance in nasal physiologic function are a free airway and free drainage. In older children the nasal septum is often at fault, and a submucous resection may be done as early as the age of 12 years without interfering with proper nasal development.

Often removal of the adenoids alone will give beneficial results, especially in young children. The tonsils should be removed if they are definitely obstructing the fauces, if they are undoubtedly infected or if there is cervical adenitis. There are many more indications, but these are the most important.

LANGMANN, New York.

THE DEAF-MUTE; A MEDICOSOCIAL STUDY. R. LABBÉ, Rev. méd.-sociale et de protec. de l'enf. **7**:1 (Jan.-Feb.); 88 (March-April); 161 (May-June); 258 (July-Aug.) 1939.

In the opinion of the author, who is chief physician of the National Institute for Deaf-Mutes in Paris, the average deaf-mute is born normal except for his auditory defect. This defect, although purely physical, brings about from the beginning a retardation of mental development which it is difficult to combat. (All degrees of native intelligence are found among deaf-mutes.) The influence of heredity, although obscure, and of family environment is more evident among deaf-mutes than among other persons.

Through education and training given the deaf-mutes in special institutions, particularly through demutization, most deaf-mutes may become able to earn their living.

The author discusses in detail the methods of instruction and demutization, vocational education, mental and character training and the historical development of schools for deaf-mutes in France. A list of such schools in France is given.

BIERMAN, Washington, D. C.

BENIGN ANGINA TONSILLARIS AND MYOCARDIAL DISEASE. G. W. PARADE, Med. Klin. **35**:269 (March 3) 1939.

The author points out on the basis of case histories of young adults that even mild tonsillar or pharyngeal infections may be complicated by myocardial damage in the form of conduction disturbances and disturbances in the electrocardiographic curve. There may be disturbance of the coronary circulation. Clinically the patient may have a slow convalescence and may suffer from dizziness, arrhythmia and anginal pain. The myocardial damage is apparently reversible, especially when the focus of infection is eliminated.

SIEMSEN, Chicago.

THE IMPORTANCE OF THE PSYCHOLOGIC FUNCTION IN THE SYNDROME OF STAMMERING. L. PATZAY-LIEBERMANN, Pract. oto-rhino-laryng. **2**:54 (Feb.) 1939.

The author discusses the results of an investigation into psychologic effects on stammering. He believes that single elements of the syndrome can be understood only if considered from a "global" standpoint. He analyzes the concomitant movements of stammerers from the standpoint of their expressional content and concludes that in the moment of stammering the entire function of expression is submitted to a dispersion and those parts of the expression which cannot be uttered verbally appear as mimic (motor) symptoms. He further analyzes the psychologic conditions which favor the occurrence of stammering.

PERSKEY, Philadelphia. [ARCH. OTOLARYNG.]

Diseases of Lungs, Pleura and Mediastinum

CONGENITAL AIR-CONTAINING CYSTS OF THE LUNG. R. S. ILLINGWORTH, Proc. Roy. Soc. Med. **32**:318 (Feb.) 1939.

The case of a girl aged 1½ years is reported. There was nothing of note in the family history. There was no history of tuberculosis. The child was taken to the hospital on account of symptoms suggestive of masturbation, dating from an attack of bronchitis seven months previously. The past history and the results of physical examination, including roentgenograms of the chest and bronchographic and bronchoscopic data, are given fully in the report. The physical signs and the roentgenograms showed no change in the child's condition during her stay in the hospital, and the signs were still present when she attended the outpatient department three months later.

WILLIAMSON, New Orleans.

CONGENITAL CYSTIC DISEASE OF THE LUNG. R. S. ILLINGWORTH, Proc. Roy. Soc. Med. **32**:602 (April) 1939.

A boy aged 3 years and 6 months had an attack of bronchopneumonia about a year before his admission to the hospital. On examination he was pale and ill looking. He was apyrexial. There were rhonchi and coarse rales at the bases of both lungs, and from the nose there was a mucopurulent nasal and postnasal discharge. Early clubbing of the fingers and toes was present. The sputum was thick, green and offensive; a culture showed hemolytic streptococci and Pfeiffer's bacillus. The Mantoux reaction was negative (1:1,000). Roentgen examination of the chest showed signs suggestive of bronchiectasis at the base of the right lung, which was thought to be due to congenital cystic disease. Roentgen examination also showed opacity of both antrums. Bronchographic examination showed collections of iodized poppyseed oil in spaces which were thought to be cysts. The sputum, examined at a later date, showed a great number of organisms but no tubercle bacilli. The tonsils and adenoids were removed and the antritis treated. Daily postural drainage was instituted. When the patient was last seen, the general condition was improving, but the condition of the chest was unchanged. The report is accompanied by roentgenograms of the lungs.

WILLIAMSON, New Orleans.

THE BITONAL COUGH IN EARLY INFANCY. A. B. MARFAN, Nourrisson **27**:133 (May) 1939.

The bitonal cough is made up of two distinctly different sounds, not like the usual cough, of one sound. One of the two sounds is low and not clearly defined; the other is high pitched and has rather a singing quality. Both sounds are produced simultaneously, but they seem to be produced by two different larynxes. Their origin is to be found in the compression of the tracheobronchial tree by enlarged lymph nodes. The underlying cause, consequently, is mainly tuberculosis. The author has exceptionally observed the bitonal cough when congenital syphilis was the cause of the enlargement of the tracheobronchial nodes, or when cardiac enlargement—due to congenital malformations—led to compression of the bronchi. He quotes autopsy records in confirmation of his statements.

HEYMAN, Cleveland.

DETERMINATION OF TYPES OF PNEUMOCOCCI ISOLATED FROM INFANTS WITH BRONCHOPNEUMONIA ACCORDING TO THE CLASSIFICATION OF COOPER. A. POUCHÉ, Med. ital. **20**:415 (Aug.) 1939.

The author typed the pneumococci obtained from infants up to 2 years of age who were afflicted with bronchopneumonia. He used agglutinating serum for the types from the Lederle Laboratories in New York. From the 51 cases fourteen different types were found. Types XIV and VI were the commonest. Type I was found in 5 cases, type III in 3 cases, type IV in 2 cases, type V in 1 case, type VI in 8 cases, type XI in 1 case, type XII in 4 cases, type XIII in 3 cases, type XIV in 9 cases, type XVIII in 5 cases, type XIX in 5 cases, type XXII in 1 case, type XXIII in 2 cases and type XXIX in 2 cases.

MARTOCCIO, Utica, N. Y.

GUANIDINEMIA IN ACUTE INFECTIONS OF THE RESPIRATORY TRACT OF CHILDREN. A. M. DORDI, Riv. di clin. pediat. **37**:448 (May) 1939.

Observations were made on the guanidine of the blood of 37 children with pneumonia or empyema. The guanidine in the blood was found to be elevated in those with hepatitis as indicated by enlargement of the liver. The appearance of hyperguanidinemia was ordinarily an unfavorable prognostic sign.

HIGGINS, Boston.

VALUE OF REPEATED TESTS AT INTERVALS OF SEVERAL YEARS IN SYSTEMATIC ROENTGEN EXAMINATION OF THE CHEST OF CHILDREN. M. TEPPATI, Riv. di clin. pediat. **37**:481 (June) 1939.

The thoracic roentgenograms of 27 children were compared with similar pictures taken five years previously. Of 9 reported as showing tuberculosis at the earlier examination, 2 showed spread, 2 diminution and 5 no change in the lesion. Of 5 previously reported "negative," all were still "negative"; of 13 reported doubtful, 7 showed diminution of the signs and 6 no change.

HIGGINS, Boston.

CONNECTIONS OF THE PLEUROPULMONARY LYMPHATIC SYSTEM AND THEIR CLINICAL IMPORTANCE. S. FOLENA, Riv. di clin. pediat. **37**:804 (Sept.) 1939.

An anatomic discussion is presented of the lymphatics of the lungs and pleura and of their connections to the supraclavicular, the subdiaphragmatic and the axillary glands. Knowledge of these connections may prove an important aid in the early diagnosis of tuberculosis.

HIGGINS, Boston.

PRODUCTION OF PNEUMONIA AFTER VARYING PREPARATORY TREATMENTS. H. REPLOH and H. EICKHOFF, Arch. f. Hyg. **122**:212 (June) 1939.

The authors were able to produce in mice characteristic pulmonary changes of lobar distribution and containing typical cells by infection with pneumococci administered by inhalation with the animals under ether anesthesia three weeks after a preparatory treatment with killed pneumococci.

Killed pneumococci administered by inhalation with the animals under ether anesthesia or by intravenous injection furnished the most impressive results. In general, septicemia and pneumonia excluded each other; in instances of outspoken findings in the lungs, culture of the blood was sterile, but the mice died of septicemia when there were no changes in the lungs.

FALKENHEIM, Rochester, N. Y.

PNEUMOCOCCUS EMPYEMA TREATED BY LOCAL IRRIGATION WITH SULFONAMIDE. TE-LUNG KUO, Chinese M. J. **56**:155 (Aug.) 1939.

The author reports the case of a child aged 13 months who was treated for subacute pneumococcus empyema through a trocar in the chest. After drainage of the pus the cavity was washed with 200 cc. of 1:1,000 sulfonamide in physiologic solution of sodium chloride; in addition, 50 cc. of solution was left in the chest. Three days later only a small amount of reddish fluid was found, which proved to be sterile. A few subsequent punctures of the chest and roentgen examinations showed that the fluid had been promptly absorbed and the lung fully expanded. The author attributes this satisfactory result to local treatment with this drug.

NEFF, Kansas City, Mo.

A CASE OF AGENESIS OF THE RIGHT LUNG. T. HEEMSTRA, Nederl. tijdschr. v. geneesk. **83**:2750 (June 10) 1939.

Heemstra describes a case of agenesis of the right lung, diagnosed during life. The infant died when 6 weeks old.

VAN CREVELD, Amsterdam, Netherlands.

TWO CASES OF EMPHYSEMA OF THE MEDIASTINUM. M. DYKSTRA, E. GORTER, A. GYSBERTI HODENPYL and A. VON WESTRIENEN, Maandschr. v. kindergeneesk. **8**:306 (May) 1939.

1. A boy of 8 years had been exposed during a short time to inhalation of ammonia. There was no subcutaneous emphysema, but the air had accumulated between the heart, the diaphragm and the left mediastinal pleura, as was found on roentgen study. This localization of the emphysema and the absence of the subcutaneous emphysema can be explained by the fact that the air had gotten into the left part of the anterior mediastinum, which found a rather isolated space. The boy recovered.

2. A girl of 1½ years had mediastinal emphysema after an attack of dyspnea (probably an attack of asthma); there was also extensive subcutaneous emphysema. This child also recovered. The cause of the mediastinal emphysema is discussed. The therapy could be only symptomatic.

VAN CREVELD, Amsterdam, Netherlands.

Diseases of the Gastrointestinal Tract, Liver and Peritoneum

VITAMIN B_1 AND LIVER EXTRACT IN THE TREATMENT OF NON-SPECIFIC CHRONIC DIARRHEA AND COLITIS. G. CHENEY, Am. J. Digest. Dis. **6**:161 (May) 1939.

Thirty-two persons suffering from nonspecific chronic diarrhea and colitis were treated with vitamin B_1, with liver extract or with both vitamin B_1 and liver extract. From this study the following conclusions were arrived at:

1. Patients with chronic diarrhea of unknown cause may respond almost specifically to the administration of vitamin B_1. Most of them will improve on oral therapy alone, but some may require treatment by injection.

2. A number of patients suffering from constipation predominantly of the atonic type, occasional short bouts of diarrhea, mucus in the stools and varying degrees of abdominal distress, gas and general exhaustion (mucous colitis) respond favorably to treatment with vitamin B_1. The majority of these patients require to be treated parenterally, but some will show improvement on oral therapy alone.

3. The majority of patients with idiopathic ulcerative colitis will have a remission during the first month of treatment with concentrated liver extract, administered parenterally. This is borne out by the improvement which occurred in 18 of 19 persons so treated. The improvement is not only symptomatic; the characteristic ulcerations of the bowel tend to heal.

4. The 32 patients with chronic diarrhea and colitis who were treated with vitamin B_1 and with liver extract were ordinarily not cured, as exacerbations usually occurred after the treatment was stopped.

5. The effectiveness of liver extract in the treatment of ulcerative colitis is apparently not due to vitamin B_1 or to vitamin B_2 but to some as yet unidentified substance contained in the extract. It seems probable that this substance improves the resistance of the colon to infection.

MORRISON, Savannah, Ga.

ROENTGENOLOGICAL ASPECTS OF THE CONGENITALLY SMALL COLON AND OF INTESTINAL OCCLUSIONS. HOWARD B. HUNT, Am. J. Roentgenol. **41**:564 (April) 1939.

The author presents a review of the literature on the subject. The embryology, pathology and clinical manifestations are briefly discussed, and 5 cases of congenital microcolon are presented. Microcolon is interpreted as secondary to atresia or stenosis of the bowel proximal to the narrowed colon; the more complete the obstruction, the smaller the colon. Atresia and stenosis are considered to result from arrest or delay in normal canalization of the intestinal lumen, which, according to Lewis, usually becomes partially occluded by irregular proliferation of cells between the fifth and the tenth weeks of embryonic life. The site of occlusion in complete congenital obstruction of the small intestine can usually be determined by the plain film. No barium is given orally. A barium sulfate enema, however, is safe if it is followed by an oil enema of equal volume, and is often a helpful diagnostic procedure. The prognosis with proper surgical repair is good for congenital obstructions of the duodenum and very poor for obstructions of the jejunum and ileum unless the latter are due to peritoneal bands or to volvulus. Bouslog has pointed out that the jejunum of the infant fails to show the "feathery" appearance of the adult types, and the distended jejunum in his cases failed to show the "herring-bone" pattern evident in the distended adult jejunum.

SQUIRE, Chicago.

ADYNAMIC ILEUS OF THE NEWBORN. JAMES B. GILLESPIE and J. C. THOMAS ROGERS, Arch. Pediat. **56**:269 (May) 1939.

The case of a boy is reported.

ORR, Buffalo.

ACUTE DEHYDRATION CAUSED BY INTESTINAL INFLAMMATION IN AN INFANT. JULIUS SURANYI and ROBERT MARTIN, Arch. Pediat. **56**:574 (Sept.) 1939.

A 14 month old infant died of diarrhea and vomiting lasting ten days. Severe signs of dehydration existed. Colon bacilli were found in the vomitus. The pathologic changes were confined to the lower part of the duodenum and the entire length of the jejunum, where signs of severe inflammation were present. Microscopic examination of the segments of the intestines mentioned showed that the upper layer of mucosa and the crypts of Lieberkuhns were widely necrotized. Numerous colon bacilli were observed on the mucosa and between the villi.

ORR, Buffalo.

DIAPHRAGMATIC HERNIA IN INFANTS. E. M. MILLER, A. H. PARMELEE and H. N. SANFORD, Arch. Surg. **38**:979 (June) 1939.

The authors report 2 cases in which diaphragmatic hernia was diagnosed in early infancy. Both the patients were operated on. One infant made a complete recovery; the other died. It is pointed out that few infants live long if obstructive sypmptoms develop. Successful operative results in infants under 1 year of age are few. A survey of the literature up to 1936 showed that only 8 infants survived out of 17 infants who had been operated on.

KAISER, Rochester, N. Y.

THE "ACUTE ABDOMEN" IN CHILDREN. JESSE R. GERSTLEY, M. Clin. North America **23**:139 (Jan.) 1939.

The pediatric diagnostician knows that almost any condition in the field of medicine may present symptoms referable to the abdomen. He knows also that in view of the serious nature of certain of these conditions he must be clear and prompt in forming conclusions. For purposes of diagnosis, a scrupulously accurate history is almost more important than a physical examination. It is necessary in history taking not only to determine the mode of development of the present illness but to investigate carefully such antecedent possibilities as trauma, sensitiveness to foods, bleeding, purpura and rheumatism and other acute infections.

Gerstley points out that in childhood the symptoms of appendicitis may not develop in the usual chronologic order, although the pain always starts in the abdomen. Elicitation of superficial tenderness by light touch is important, and rigidity is best brought out by this maneuver. Tenderness, however, may require rather deep palpation. Tenderness and rigidity are always located at the site of the appendix. In making a rectal examination, the left side should be palpated first. The use of a suppository, by causing expulsion of gas, may clear up a puzzling case. In all doubtful cases roentgen examination of the chest should be made, since pneumonia may give predominantly abdominal symptoms. Should the history and physical examination favor a diagnosis of appendicitis, operation should not be deferred because the white blood cell count is normal. In differential diagnosis, certain rather rare conditions should be kept in mind, such as Meckel's diverticulum, acute mesenteric lymphatic adenitis and migratory peritonitis. If after a reasonable time the diagnosis remains in doubt, operation should be performed.

H. SMITH, Ogden, Utah.

SURGICAL ASPECTS OF THE ACUTE ABDOMEN IN INFANCY AND CHILDHOOD. CHARLES W. MCLAUGHLIN JR. and HERBERT H. DAVIS, Nebraska M. J. **24**:304 (Aug.) 1939.

Acute abdominal emergencies in children, although they offer a lesser variety of lesions than do the same diseases in adults, are more difficult of diagnosis. As the evidence of disease is objective rather than subjective and the history must be taken from an observer rather than the patient, problems of diagnosis are more confusing. The order of onset of symptoms of appendicitis, the most frequent abdominal lesion in childhood, is the same as in the adult: (1) pain, (2) nausea and vomiting, (3) tenderness, (4) fever and (5) leukocytosis. Abdominal rigidity indicates peritoneal irritation. The average temperature in acute appendicitis is 100.4 to 101 F., and the average white cell count is about 15,000 per cubic centimeter of blood. Perforation occurs with rapidity because of the large amount of lymphoid tissue, the thin-walled appendix and an underdeveloped omentum. In approximately 95 per cent of children under 5 years of age the process will be sufficiently advanced to require drainage. The mortality in all cases in infancy and childhood

approaches 15 per cent. Three factors are responsible for the high mortality: (1) difficulty of diagnosis in the early stage; (2) rapidity of progress, and (3) administration of purgatives.

In pyloric stenosis a tumor can be palpated in less than 50 per cent of the cases. Local anesthesia is preferable; 5 cc. of a 0.5 per cent solution of procaine hydrochloride has been found satisfactory. As with all surgical procedures in children, the water balance must be maintained, and the help of a pediatrician is of great assistance.

For pneumococcic peritonitis the recent trend is toward the conservative side as regards operation. Intussusception, mesenteric adenitis, purpura and diverticulitis must be kept in mind.

HAMILTON, Omaha.

ENTEROGENOUS CYSTS WITH REPORT OF A CASE INVOLVING THE TERMINAL ILEUM. M. B. DOCKERTY, R. L. J. KENNEDY and J. M. WAUGH, Proc. Staff Meet., Mayo Clin. **14**:664 (Oct. 18) 1939.

A case of an enterogenous cyst obstructing the terminal portion of the ileum in an 8 month old infant is reported. Recovery followed removal. Such cysts, which are congenital, arise from and reproduce the structures of the normal intestinal wall.

MILLER, South Bend, Ind.

EPIDEMIC OF NURSERY DIARRHEA. J. P. COSTELLO and H. E. LIND, South. M. J. **32**:620 (June) 1939.

Costello and Lind report an epidemic of nursery diarrhea which was similar in many respects to those reported in various hospitals throughout the temperate zone. They feel that the severe toxemia was a result of absorption of toxins from the development of bacteria in the gastrointestinal tract.

They realize fully that Proteus vulgaris may be found in healthy persons but that it is rarely found predominantly in the intestinal tract, and rarely, if at all, in the stomach, except under pathologic conditions.

The disease was characterized by its constantly severe and frequently fatal toxemia. Its onset, cause and duration were so similar in these cases as to suggest strongly that this disease should be classified as communicable.

SCHLUTZ, Chicago.

CHRONIC DYSPEPSIA IN EARLY AND LATE PERIODS OF CHILDHOOD. S. MEKLER, Arch. de méd. d. enf. **42**:282 (May) 1939.

Dilatation of the stomach is as common a causative factor in digestive disturbances of children, the author asserts, as in those of adults. It is occasionally associated with aerophagia.

In a report of 87 autopsies made by Comby and Rousseau on infants up to 2 years of age, it is stated that 64 showed gastric ectasia of considerable degree. Symptoms in these cases were variable and often suggested involvement of the nervous system or of the circulatory apparatus rather than involvement of the gastrointestinal tube. When proper regulation of the diet is made in such cases, the symptoms disappear. Gastric analysis is of little value, as the variations in health are so pronounced. From whatever cause, it must be remembered that young subjects are particularly susceptible, in the rapid evolution of the organism, to serious damage from gastrointestinal disturbances, resulting, as they frequently do, in a retardation of nutrition and growth from defective absorption. The importance of dental defects and of intestinal parasites is noted.

AMESSA, Denver.

Nervous Diseases

RESPIRATORY METABOLISM OF INFANT BRAIN. H. E. HIMWICH, Z. BAKER and J. F. FAZAKAS, Am. J. Physiol. **125**:601 (March) 1939.

The data presented indicate that the respiratory processes of the infant brain, like those of the adult brain, are dependent on added carbohydrate. Brains of infant rats (form 1 to 24 days old) were used. The metabolic rate was lower in the infant brains, but the difference was quantitative rather than qualitative and correlated with the difference in concentration of protein, which is lower in the infant brain.

NOURSE, Cleveland.

INADEQUACY OF THE EVIDENCE FOR HEREDITARY PREDISPOSITION IN EPILEPSY. E. ZISKIND and E. SOMMERFELD-ZISKIND, Am. J. Psychiat. **95**:1143 (March) 1939.

After discussing at length the evidence favoring a hereditary predisposition to the development of epilepsy, the authors proceed to analyze the various factors and concepts of the syndrome. They conclude that the theories in favor of a hereditary factor are far from convincing and that those against the inheritance of the condition seem more valid. The fact that so many persons with epilepsy point to some one in the family background who had convulsive seizures indicates that the patient simply has a better knowledge of his family history than the nonepileptic person has of his, and in the view of the authors the history is not of great significance.

JAHR, Omaha.

TERATOMA OF THE BRAIN. J. ZISKIND and H. J. SCHATTENBERG, Arch. Pediat. **56**: 347 (June) 1939.

True instances of teratoma of the brain are very rare, and in those that have been reported in the literature the teratoma occurred near or at the midline. In the case reported by the authors the teratoma involved the right cerebral hemisphere, filling a considerable portion of the right lateral ventricle and invading the region of the diencephalon.

ORR, Buffalo.

FAMILIAL DIFFUSE SCLEROSIS (PELIZAEUS-MERZBACHER DISEASE). KARL O. VON HAGEN and C. W. SULT JR., Bull. Los Angeles Neurol. **4**:23 (March) 1939.

Von Hagen and Sult report observations on 2 brothers with Pelizaeus-Merzbacher disease, including the pathologic changes in 1 of them. The first patient, a boy aged 9 when examined, had had a normal birth, also normal development until he was 4½ years old. At that time he began to walk stiffly on his toes. Spastic paraplegia progressed steadily; in two years he was bedfast. Speech was gradually lost. Examination showed extreme spastic paraplegia, widespread athetotic movements and frequent extensor spasms with upward deviation of the eyes. Optic atrophy was present bilaterally, and the plantar responses were pathologic. Hearing was preserved, and there were no gross sensory disturbances. There was marked mental defect. Laboratory studies, including the Wassermann test of the blood, gave negative results.

Pathologically, there was widespread demyelination of the white matter except for the arcuate fibers and some islands of preserved myelin. The areas of demyelination were essentially in the form of perivascular rings. The white matter of the cerebellum was similarly involved, while the basal ganglions, dentate nuclei, cerebellar peduncles and medullary nuclei were well preserved. The cells of the cortex were normal, but the projection fibers were largely demyelinated. There were descending degeneration of the pyramidal tracts and incomplete demyelination of the extrapyramidal tracts and the ground bundles in the cord. Some astrocytic gliosis was present, but the glia was undergoing degeneration with loss of processes and formation of "ameboid glia."

The brother was normal until the age of 3½ years, when he began to lose the power of speech. Movements became awkward and the patient apathetic. The pupils did not react to light; the tendon reflexes were increased, and Babinski and Oppenheim reactions were present on the left. Sensation was normal. Von Hagen and Sult believe that the process began in the white matter of the cerebrum and cerebellum, with secondary degeneration in the spinal cord. The involvement of the extrapyramidal and ground bundle pathways in the cord must, however, have been primary. MACKAY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

EPIDEMIC ENCEPHALITIS TYPE B IN CHILDREN: CLINICAL OBSERVATIONS IN THE 1937 OUTBREAK. P. J. ZENTAY and J. BASMAN, J. Pediat. **14**:323 (March) 1939.

Zentay and Basman summarize the clinical observations made on 24 children who were observed during the summer of 1937, in the second epidemic of encephalitis type B to occur in the area of St. Louis. The incidence was highest among the children aged 6 to 10 years. Five of the children were under 1 year of age, and 1 child was only 4 months old. The onset was usually sudden and was preceded in the majority of the children by symptoms of a mild infection of the upper respiratory tract. The prodromal stage lasted from twenty-four to seventy-two hours, after which the central nervous system was invaded. The children suffered from intense headaches, which continued until a disturbance of consciousness led sooner or later to a state of somnolence or deep stupor. At this time the face of the patient assumed a blank staring expression. There was nothing characteristic in the temperature curve. The disturbance of consciousness lasted from a few hours to from fifteen to twenty days. A change of personality and of behavior accompanied the state of somnolence or stupor and often continued beyond the acute stage of the disease. No follow-up studies have as yet been made to ascertain whether any permanent change of personality remained in any of the patients. Signs of meningeal irritation were present in all patients. Convulsions of the tonicoclonic type occurred in 8 patients. In 7 patients mild transient muscle paralyses in the form of monoparesis or hemiparesis occurred, accompanied by the usual signs of a lesion in an upper motor neuron. The lesions lasted only a few days and cleared up completely. Changes in reflexes were noted only with the transient pareses. The abdominal reflexes were absent during the stage of meningeal irritation. The cerebrospinal fluid was under increased pressure. The average cell count was 110 and the mean cell count 70, the highest count occurring between the fourth and the eighth day after onset. Early in the disease polymorphonuclear cells predominated, with a gradual shift to predominance of mononuclear cells. The Pandy test showed increase in protein in about 60 per cent of the children. All of the children except a previously defective epileptic child recovered. No residual signs were noted. RAUH, Cincinnati.

PACHYMENINGITIS INTERNA HAEMORRHAGICA. E. HINDEN, Proc. Roy. Soc. Med. **32**:1375 (Sept.) 1939.

A boy of 8 months had been normal except that for one month before admission to the hospital he had been subject to transient fits of slight intensity. He was admitted to the hospital because of a prolonged severe convulsion that morning, followed by vomiting. He looked ill and was very white, cold and limp. The fontanelle was large and tense. The pupillary reaction to light was absent, and there were massive hemorrhages into both retinas. The disks were not seen. The temperature was 101 F. Moist rales were heard in both lungs. Lumbar puncture revealed the cerebrospinal fluid under increased pressure; otherwise the fluid was normal. The temperature fell after five days, when the symptoms commenced to clear up. The combination of mild hydrocephalus, convulsions and retinal hemorrhages suggested the diagnosis. Puncture of the fontanelle was performed twice, the first time with negative results; the second time a clear brown fluid welled out of the needle. WILLIAMSON, New Orleans.

ACUTE NONSUPPURATIVE ENCEPHALITIS FOLLOWING PNEUMONIA. GRACOSKI and MARCULESCU, Bull. Soc. de pédiat. de Iasi **9**:3, 1938.

In a 3 year old child, on the sixth day of convalescence from pneumonia, a convulsion suddenly developed, and the child became profoundly prostrated. There were paralysis of the left facial nerve and contractures of the extremities, which gradually improved. The spinal fluid contained 13 lymphocytes per cubic millimeter and a low amount of sugar. The prognosis was thought to be good.

LESLIE, Evanston, Ill.

MEDULLOBLASTOMA: A CASE IN WHICH AN OPERATION WAS PERFORMED AND TEMPORARY IMPROVEMENT OBTAINED. GRACOSKI, A. MORUZI and BRIESE, Bull. Soc. de pédiat. de Iasi **9**:6, 1938.

A ventriculographic examination showed blockage between the third and the fourth ventricle. The tumor could be only partly removed at operation, but with repeated roentgen therapy definite clinical improvement occurred. Unfortunately, contact with the patient was lost.

LESLIE, Evanston, Ill.

POSTGRIPPAL MYOCLONIA. GRACOSKI, HURMUZACHE and MARCULESCU, Bull. Soc. de pédiat. de Iasi **9**:11, 1938.

This case of generalized clonic movements occurring after a grippal infection is the third such case to be reported by the authors. The patient was a baby 10 months old. The temperature remained elevated, and the roentgenogram showed a shadow which was interpreted as pneumonia. The cerebrospinal fluid was normal.

LESLIE, Evanston, Ill.

ACUTE TUMOR OF THE BRAIN SIMULATING ENCEPHALITIS. R. CLÉMENT, P. AUZÉPY and H. MASCHAS, Bull. Soc. pédiat. de Paris **37**:60 (Feb.) 1939.

A girl 13 years old had convulsions involving the right upper extremity, followed by right-sided hemiparesis. There was no papilledema. The child's condition became progressively worse, and she died twenty-three days after her admission to the hospital. Necropsy revealed a cerebellar tumor which had undergone cystic degeneration. It was thought to be a malignant glioma. No histologic examination was made.

BENJAMIN, Montreal, Canada.

RHINOLOGIC MENINGITIS ACTUALLY MENINGEAL LABYRINTHITIS. H. BARTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:1 (March) 1939.

The author reports an interesting case of a 7½ year old boy who a year prior to the onset of symptoms of meningeal disease had acute otitis media which cleared up shortly after tonsillectomy. Residual evidences of infection were not present.

Four weeks prior to the onset of meningeal symptoms, the patient fell while playing, struck his nose and suffered a fracture of the nasal bone, with marked deviation of the nose and septum to the right. At that time, the deformity was corrected, and the child was apparently well until the day before admission to the hospital, at which time he again fell and again struck his nose. At this time there was decided swelling of the left upper lid and the left side of the nose, extending up to the nasal bridge. This was due to a pronounced hematoma, extending up to the inner canthus of the left eye. However, roentgen evidence of fracture was not present. That evening the temperature began to rise, and the following morning the patient had projectile vomiting; he was subsequently admitted to the hospital.

Examination on admission revealed definite nuchal rigidity and Kernig's sign. The ear drums were somewhat retracted but otherwise normal. There was a large hematoma along the left side of the nose, and the septum, which appeared also

somewhat swollen, was moderately deviated to the right. Pus was not seen in the nose. The pharynx was normal. Roentgen examination of the skull did not show evidence of a fracture. The sinuses were all clear. A spinal tap was performed; the fluid was under considerable pressure, appeared cloudy and contained 8,106 cells, and the Pandy test was positive. The following day there was evidence of a septal abscess; this was incised and free pus obtained, culture of which revealed pure pneumococci. That day complete deafness developed in the left ear, with spontaneous nystagmus to the right. Another lumbar puncture was performed; the cell count ranged from 8 to 12,000 (many polymorphonuclears), but the culture was sterile. While the clinical picture was that of meningitis, presumably of rhinologic origin, some inconsistent findings made it difficult for the neurologist to localize either a central process or the site of origin. The encephalogram did not yield any additional information and was not of material aid.

In view of the continuation of the fever and persistence of the symptoms, it was finally decided that an operation should be performed on the external frontal sinus. This was done seventeen days after onset of symptoms, and it revealed a normal sinus, the mucous membrane of which was perfectly normal in appearance; the inner plate was also normal and its removal revealed a normal-appearing dura. Puncture of the subdural space gave negative results, but severe bleeding occurred, and the operation was interrupted. The child became progressively worse, and a few days later a second operation was performed. The site of the previous operation was revised. The ethmoid and sphenoid sinuses on the left side were opened widely. There was no frank evidence of pathologic change. The right sinuses also were explored, and they were normal. Four days after this operation, the child died.

Postmortem observations showed that the anterior and middle fossae were normal but that there was marked leptomeningitis in the posterior fossa, apparently arising from the left side. In addition, there was suppurative labyrinthitis, which may have accounted for the deafness in the left ear and the spontaneous nystagmus. The posterior vertical canal was eroded and filled with pus. However, the vestibule, the cochlea and the other two semicircular canals did not show any frank suppuration but only evidence of some fibrosis and a reactive process.

In his summary, the author makes the following points: A case is presented of a 7½ year old boy in whom meningitis followed an injury to the nose. The meningitis occurred simultaneously with a septal abscess. The clinical picture suggested a suppurative focus in the nose or nasal sinuses, but this was not substantiated by either operative findings or autopsy. The patient died within three weeks. Labyrinthitis developed on the left because of the meningitis. Histologically, the suppurative inflammation of the posterior semicircular canal produced osteitis of the temporal bone.

(The possibility of a latent infection in the labyrinth, as a result of old otitis media which had been activated by the recent injury, should be borne in mind.—Abstracter.)

PERSKY, Philadelphia. [ARCH. OTOLARYNG.]

Psychology and Psychiatry

MOTILITY OF SCHOOL CHILDREN DURING SLEEP. G. GIDDINGS, Am. J. Physiol. **127**:480 (Oct.) 1939.

The time spent in bodily movement during sleep by normal, healthy adults has been shown to be about half a minute per hour (Kleitman, Cooperman and Millin). The authors devised a new apparatus to measure accurately the motility during sleep of a number of subjects simultaneously. Normal children between the ages of 9 and 14 years were studied. The total length of time spent in motion during nine hours in bed amounted to less than five minutes, which is comparable to the time recorded for adults. The greatest activity occurred during the first half hour.

From the second hour onward there was a gradual increase in motility, which reached its highest point in the last hour. No sex difference was noted.

NOURSE, Cleveland.

MENTAL DISORDERS IN TRIPLETS. A. J. ROSANOFF, L. M. HANDY and I. R. PLESSET, Am. J. Psychiat. **95**:1139 (March) 1939.

This is a report on 12 sets of triplets, comprising 33 surviving and 3 stillborn children, collected in the course of a study of mental disorders in twins. The triplets were selected on the basis of at least one child in each set having some form of mental aberration. Of the 33 subjects, 17 presented definite neuropsychiatric conditions, ranging from subnormal intelligence through difficulties in child behavior, juvenile delinquency, adult criminality, psychosis and epilepsy.

JAHR, Omaha.

FINGER AGNOSIA IN CHILDREN. A. STRAUSS and H. WERNER, Am. J. Psychiat. **95**:1215 (March) 1939.

In children the inability to recognize, indicate on request, name or choose fingers of the hand is associated with a low intelligence quotient and special deficiency in number work. The tests are discussed in connection with a case report.

JAHR, Omaha.

REACTION OF PARENTS TO READING FAILURE. M. I. PRESTON, Child Development **10**:173 (Sept.) 1939.

In 100 cases of reading failure in which the children were of normal mentality (intelligence quotients, from 90 to 140) the parental reaction was found to be mainly that of increasing intolerance as the years rolled by (2 to 10), which was expressed in attitudes of annoyance, anxiety, distress, anger, disgust, mortification and desperation. In the majority of cases the parent placed the blame on the child, bringing forth acid comparisons with reading siblings as well as other relatives and neighbors, derogatory epithets, scorn, ridicule, reproaches, taunts, deprivation of privileges and physical punishment (in one third of the cases), along with never ending nagging, scolding and prodding, all especially virulent when the home felt it necessary to take on the task of teaching. These reactions caused rebellion at the injustice of the treatment; or, if the burden of guilt was accepted by the child, a disintegration of personality took place over the years as feelings of guilt overwhelmed him. Two appeals are made: (1) that experts be called in as soon as the blighting disease of nonreading appears (as devastating as neglected tuberculosis); (2) that for preventive work teachers of *beginning* reading (a highly technical process) be specialists whose intensive training under supervision has made them masters of all the skills and arts of many methods of solving a problem, not just the "get rich quick" method in vogue.

FROM AUTHOR'S SUMMARY.

THE WANDERING I. Q.: IS IT TIME FOR IT TO SETTLE DOWN? B. R. SIMPSON, J. Psychol. **7**:351 (April) 1939.

The possibility of improving the intellectual ability of children is one of fundamental importance to those interested in the growth and development of children.

This article is a bitter attack on the publications and claims of the Iowa group, which is headed by Beth Wellman. This group have claimed that children attending the University of Iowa schools undergo an increase in their intelligence quotient. Simpson charges and attempts to prove that they have misinterpreted their data and that they present no dependable evidence in support of their "glamorous claims."

WEINFELD, Highland Park, Ill.

AGGRESSION IN EARLY CHILDHOOD. KARIN STEPHEN, Brit. J. M. Psychol. **18**:178, 1939.

Stephen believes that aggression is first called forth in inner anxiety situations in infancy which seem to threaten the ego. If the frustration causing the anxiety is too great, whether inner or outer or mixed, it may be experienced as intolerable ego danger and handled by flight, projection, introjection or repression. Secondarily, these do not relieve the anxiety, and a vicious cycle is set up, with useless displays of aggression. When this mechanism is carried to adult life, it defeats healthy living. Stephen believes, however, that aggression is necessary to normal development and if properly used is valuable in helping the person to integrate properly with society.

ALLEN, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

AFTER-RESULTS OF CHILD GUIDANCE: A FOLLOW-UP OF FIVE HUNDRED CHILDREN TREATED AT THE TAVISTOCK CLINIC, 1921-1934. A. MABERLY and B. STRUGE, Brit. M. J. **1**:1130 (June 3) 1939.

Maberly and Struge review the after-results of child guidance at Tavistock clinic. These results are somewhat exhaustively analyzed by age, sex and intelligence. They conclude that the results justify the efforts.

ROYSTER, University, Pa.

THREE CASES OF PHENYLPYRUVIC OLIGOPHRENIA. R. N. BATES, J. Ment. Sc. **85**:273 (March) 1939.

Bates describes the physical characteristics of 3 patients with phenylpyruvic oligophrenia. They were boys aged from 9 to 15 years. All were imbeciles. They were "ash blondes"; the hair of 1 boy had become lighter in the last two and one-half years. There was no pubic or axillary hair. Their eyes were light blue. Examination of the fundi revealed pale optic nerve heads and a poorly developed choriocapillaris. The skin was very fair, with a marked tendency toward dermographia and susceptibility to sunburn. All reflexes were hyperactive. There was cyanosis of the hands and feet. A brief trial of vitamin B_1 therapy in 2 patients was without effect on the excretion of phenylpyruvic acid.

KASANIN, San Francisco. [ARCH. NEUROL. & PSYCHIAT.]

MENTAL ANOREXIA IN CHILDREN. J. COMBY, Progrès méd., June 17, 1939, p. 865.

Comby feels that in many cases anorexia associated with nutritional disturbances is mistakenly thought to be hypophysial cachexia (Simmond's disease). He emphasizes the advisability of early gavage feeding as well as psychiatric treatment.

LESLIE, Evanston, Ill.

PEDAGOGIC THERAPY FOR ABNORMAL CHILDREN. H. ASPERGER, Med. Klin. **35**:943 (July 14) 1939.

Persons considerably under average intelligence, especially psychopathic ones, can be guided so as to be useful members of society. Desirable traits can be developed and poor traits discouraged. Persons with marked physical and mental sensitivity, who are thereby plunged into agonies and conflicts, are frequently endowed with capacities for experiences and accomplishments which are inaccessible to more primitive and robust persons. Proper placement is important.

In many maladjusted persons a cardinal finding is physical, mental or emotional infantilism, alone or in combination. Frequently the delay in maturation necessitates continuation of training beyond the usual time.

Two patients who were observed several years are reported on in detail.

SIEMSEN, Chicago.

Diseases of the Ductless Glands; Endocrinology

LOCAL LIPOID ATROPHY AND LIPOID DYSTROPHY. B. ALPERT and E. A. FERGUSON, Endocrinology **24**:741 (May) 1939.

Over a period of seven years the authors observed 430 insulin-treated patients. In 7.4 per cent local lipoid atrophy developed. There was no involvement of muscle. Atrophic areas are insensitive to pain, and it is suggested that such sites should be used for injections inasmuch as frequent changing of site can lead to multiple areas of atrophy.

JACOBSEN, Buffalo.

CLINICAL USE OF SYNTHETIC MALE SEX HORMONE [ANDROGEN]. H. H. TURNER, Endocrinology **24**:763 (June) 1939.

Of 54 patients treated with a synthetic androgen (testosterone propionate), 8 were children of ages varying from 7 to 16 years—all boys showing deficient genital development. All responded promptly to treatment by increased growth of the penis and development of more masculine features. No untoward by-effects were noted. No changes in bones as seen in roentgenograms could be demonstrated. Much was accomplished with respect to the mental attitude of the patients.

JACOBSEN, Buffalo.

COMPARATIVE POTENCY OF COMMERCIAL ANTERIOR PITUITARY-LIKE [GONADOTROPIC] PREPARATIONS. F. GAENSBAUER and J. T. BRADBURY, Endocrinology **24**:867 (June) 1939.

Five commercial preparations of the gonadotropic factor in the urine of pregnant women were assayed by a common method to determine their comparative potency. It is concluded that antuitrin S and follutein are approximately twice as potent as antophysin, APL and pregnyl.

JACOBSEN, Buffalo.

INFLUENCE OF THYROID HORMONE ON SERUM PHOSPHATASE. N. B. TALBOT, Endocrinology **24**:872 (June) 1939.

The level of phosphatase in the serum is believed to be an index of osteoblastic activity and as such is related to bone formation. Hypothyroidism during childhood results in retarded osseous development. Thyroid therapy stimulates bone growth.

As was to be expected in view of these facts, it was found that the serum phosphatase of untreated hypothyroid children tends to be abnormally low (average 3.0 Bodansky units) and that it is restored to normal levels (average 8.5 Bodansky units) by thyroid therapy.

JACOBSEN, Buffalo.

DIABETES MELLITUS IN CHILDHOOD AND YOUTH. P. White, Practitioner **142**:576 (May) 1939.

The apparent difficulty in the management of juvenile diabetic patients has in general two explanations: 1. The disease is uncommon in childhood. Therefore, unless the physician is especially interested in diabetes, he sees few children afflicted with it, and consequently he lacks experience. 2. Since the commonest age of onset for juvenile diabetes is that of 12 years, the child is rarely treated by the pediatrician, whose mind should be unbiased, but more often by the general practitioner or physician who, accustomed to deal with diabetic adults, expects to find the same mental attitude in the young patient and applies the same standards for control and the same principles of treatment. For this reason White points out certain features which distinguish juvenile from adult diabetes. Even the type of onset is characteristic, for in the child the disease is acute and virulent. Often the first recognition is made in the chemical coma, and the younger the patient the more striking is this fact.

The exact methods employed in the George F. Baker Clinic are outlined. The caloric requirement is based on surface area. Since in childhood this follows age closely, calories may be prescribed accordingly—1,000 to an infant of 1 year, with 100 calories being added for each year of life. For girls a maximum of 2,200 calories is reached at the age of 13. As soon as possible after maturity, the caloric prescription for girls should be reduced to avoid the obesity common in female adolescents. The maximum caloric diet prescribed for boys is 2,800, reached at 19 years of age.

The qualitative prescription, the ratio of carbohydrate to fat, is selected according to one of two schools of thought. Those students of diabetes who believe that stimulation of the pancreas results in alleviation of diabetes prescribe carbohydrate-rich and fat-poor diets. Those who believe rest of the pancreas alleviates the disease prescribe low carbohydrate diets.

All diabetic children require insulin and should be given it, preferably continuously, from the day of recognition of the disease. Three years' experience with protamine insulin leads to the conclusion that it is the ideal form to use in treatment of the juvenile disease. But, unlike experience with adults, which shows that over 50 per cent require no regular insulin in addition to protamine insulin, it is found that 90 per cent of children will require both regular and protamine insulin, which are given in separate injections simultaneously before breakfast.

GELSTON, San Francisco.

Diseases of Muscles, Bones, Joints and Lymph Glands

ANOMALIES AND VARIATIONS IN THE NORMAL SKULL FROM A ROENTGENOLOGICAL STANDPOINT. CHARLES WADSWORTH SCHWARTZ, Am. J. Roentgenol. **42**:367 (Sept.) 1939.

This is a discussion of numerous conditions in the normal skull which might be confused with pathologic processes.

The diploe vary in thickness and may present a honeycombed appearance, either local or generalized. This may be confused with hyperparathyroidism or pituitary basophilism. Variations in vascular channels are innumerable. Double suture lines reveal on close inspection a suture line of the inner table and a more serrated suture line of the outer table. Patent parietal foramens occur on either side of the midline. A calculus in the anterior lobe of the pituitary gland is apparently normal. The malar bone may be divided by a horizontal suture which is mistaken for a fracture. Other anomalies are a missing superior orbital plate of one or (rarely) both sides and underdevelopment of one or more sphenoid wings. An uncommon anomaly named *"Luckenschadel"* is described. The term means "marked with furrows or channeled" and is a hypoplastic condition of bone.

SQUIRE, Chicago.

CHLOROMA IN CHILDREN. F. R. B. ATKINSON, Brit. J. Child. Dis. **36**:18 (Jan.-March) 1939.

Chloroma is a rare disease characterized by the formation of greenish tumors, especially of the face and skull, and a leukemic blood picture. Exophthalmos, often followed by blindness, general weakness, anemia, hemorrhages, enlarged lymph nodes and enlarged liver and spleen, is an additional feature.

Two forms of chloroma are usually described, lymphatic and myeloid.

In addition to the bones of the skull, the sternum, ribs, vertebrae, femur, pelvis, tibia, humerus, wrist, clavicle and fibula have been repeatedly involved, in this order of frequency. The lymph nodes are often invaded by the tumor.

The author has been able to collect 130 cases of chloroma in children. Chloroma is more frequent in boys than in girls and is commonest in children aged from 4 to 5 years. The prognosis is uniformly bad.

LANGMANN, New York.

STILL'S DISEASE. F. R. B. ATKINSON, Brit. J. Child. Dis. **36**:100 (April-June) 1939.

The author was able to collect from the literature 86 cases in which the triad of signs described by Still was present. Atkinson states that the knees and elbows are generally attacked first, then the hands, feet, fingers and toes. The glands within the region of the affected joints enlarge. He states the belief that there are cases of Still's disease in which the spleen is not enlarged.

No new light is shed on the etiology of the condition.

LANGMANN, New York.

DENSITY OF BONE. H. A. T. FAIRBANK, Brit. J. Surg. **27**:1 (July) 1939.

The author discusses in a general fashion osteoporosis as it exists in osteogenesis imperfecta, scurvy, rickets, osteomalacia, celiac disease, renal rickets, hyperparathyroid disease, senile osteoporosis and a number of other less common conditions. These are linked in discussion with an equal list of conditions which produce increased density of bone. The author points out that both osteoporosis and osteosclerosis may be present in the same case or even in the same bone. He suggests that "marble bones" with limited distribution, sometimes known as melorheostosis, is a particular type of diffuse fibrosis of the bone. He also shows that there is some evidence that Albers-Schönberg disease may have a similar pathologic picture. He refers to the possibility that the parathyroids may be responsible for such diseases as renal rickets and "marble bones" as well as for the fibrocystic condition of hyperparathyroidism.

KEITH, Toronto, Ontario.

MULTIPLE EPIPHYSIAL DYSPLASIA. D. E. YARROW, Proc. Roy. Soc. Med. **32**: 315 (Feb.) 1939.

A boy aged 7 years had always been undersized for his age. For two years he had complained of pains in the lower limbs. Thyroid had produced no improvement. He was 7 inches (17.7 cm.) underheight and 9 pounds (4.1 Kg.) underweight. Genu valgum with 1½ inches (3.7 cm.) separation at the ankles was present, and the child had a peculiar gait. Roentgen examination showed (*1*) general decalcification, especially in the diaphyses, and deformed, stippled epiphyses with delayed growth; (*2*) irregularity and interference with the growing ends of all the long bones, and (*3*) similar changes in the ossific centers in the carpus, the tarsus and the spinal vertebrae. Enchondral, endosteal and periosteal bone formation were all affected. Other investigations gave negative results. The report is accompanied by roentgenograms of the long bones and a photograph of the patient.

WILLIAMSON, New Orleans.

OSSEOUS DYSTROPHY. DAVID LEVI, Proc. Roy. Soc. Med. **32**:601 (April) 1939.

A boy aged 12 years was taken to the hospital on account of injury to the fingers, which were deformed. The phalangeal joints were very mobile, and the fingers could be pulled into hyperextension. The Wassermann reaction was negative. Roentgenograms showed bilateral changes in the middle phalanges of the second, third and fourth fingers and both extremities of the diaphysis of the first metacarpal bone of the thumb. Both femoral necks showed epiphysial deformities. The report is accompanied by roentgenograms of affected bones. There were no changes in the shoulders or in the skull.

WILLIAMSON, New Orleans.

ACUTE OSTEOMYELITIS WITH BACTEREMIA AND STAPHYLOCOCCIC SEPTICEMIA. R. DEBRÉ and J. LEVEUF, Presse méd. **47**:361 (March 8) 1939.

It is important to distinguish between acute osteomyelitis and staphylococcic septicemia, since the treatment is entirely different. Staphylococcic septicemia

should not be treated by surgical intervention even if a metastatic focus in some bone simulates osteomyelitis. Treatment must be wholly medical.

In cases of true osteomyelitis, surgical intervention must not be premature. By immobilization of the affected bone in a plaster cast the process can be prevented from spreading, and the natural defenses of the body will wall off the infection. When frank suppuration appears, the abscess can be incised and the diseased portion of bone removed.

DAFFINEE, Boston.

ROENTGEN STUDIES OF THE SKULL IN OBESE CHILDREN. P. NOBÉCOURT and J. HAGUENAU, Presse méd. **47**:437 (March 22) 1939.

A study of 31 cases was reviewed for roentgen changes in the skull. In only 2 were there any notable abnormalities. The authors conclude that in most cases of obesity there is no pituitary insufficiency.

DAFFINEE, Boston.

ACUTE OSTEOMYELITIS OF THE HIP IN INFANTS. A. MAGRASSI, Lattante **10**: 99 (March) 1939.

Magrassi reviews his experience with 16 cases of hematogenous osteomyelitis of the hip. Pneumococci were the causative organisms in nearly every case. The focus was in the femur in all but 1 case, in which it was in the acetabulum. There were 2 deaths.

He recommends early opening of the joint and drainage of the pus to relieve the general septic reaction and to prevent dislocation of the hip. He advises traction at first and later immobilization in plaster; he recommends serotherapy and chemotherapy.

HIGGINS, Boston.

Skin Diseases; Allergy

ANAPHYLAXIS AND ALLERGY. C. A. DRAGSTEDT, Ann. Int. Med. **13**:248 (Aug.) 1939.

Experiments show that cellular injuries resulting in the liberation of histamine and peptone produce reactions virtually indistinguishable from those occurring in acute anaphylactic shock. The possibility that allergic reactions seen clinically may be produced by these nonprotein substances is suggested.

READING, Galveston, Texas.

ERYTHEMA NODOSUM IN CHILDHOOD ASSOCIATED WITH INFECTION BY OIDIUM COCCIDIOIDES. J. E. THORNER, Arch. Pediat. **56**:628 (Oct.) 1939.

Erythema nodosum occurring in 7 children ranging in ages from 3½ to 13 years was proved to be associated with Oidium coccidioides and was independent of either tuberculous or rheumatic infection.

In all 7 cases the spore form of O. coccidioides was found in the stomach washings and sputum, as verified by inoculation of guinea pigs. Cases in which material taken from the inoculated animals failed to yield the characteristic mold on Sabouraud's medium have not been included here. However, 9 other instances have been collected by the author since January 1938 in which stomach washings from children with erythema nodosum were reported positive for the fungus up to reculture stage but negative thereafter. In each instance a definite pulmonary complication accompanied the erythema nodosum, as shown by clinical and roentgen findings, but it resolved entirely within a month and failed to progress later into granulomatous or caseous lesions.

No tubercle bacilli were ever isolated from any of the specimens, and in all but a single case the Mantoux test was negative on routine examination.

In most instances presence in dusty areas or engagement in dusty activities had preceded the illness by a few weeks, permitting the inhalation of a greater

number than normal of the chlamydospore-containing mycelia from the surrounding dust. This bears out Ophul's original contention that the primary infection with Coccidioides immitis in the majority of cases occurs through the respiratory tract.

ORR, Buffalo.

IMPETIGO CONTAGIOSA COMPLICATED BY ACUTE NEPHRITIS. S. H. SILVERS, New York State J. Med. **39**:1093 (June 1) 1939.

Acute glomerular nephritis followed generalized impetigo in a 17 year old white school boy. He made a good recovery.

AIKMAN, Rochester, N. Y.

INFANTILE ACRODYNIA. J. BONABA and P. C. BLACH, Arch. de pediat. d. Uruguay **10**:483 (Aug.) 1939.

Infantile acrodynia is reported in a child 3½ years old. The infant was reared in the country. At 2 years of age, following an attack of pyelitis, peculiar changes in the nervous system were observed. The child became irritable and depressed, showed anorexia and had marked insomnia. Following this there was pain in the extremities, marked itching of the palmar-plantar surfaces and profuse sweating. The intelligence remained entirely normal. Marked cyanosis and infiltration of the skin of the hands and feet developed. Sudamina was general over the trunk, and there was marked conjunctivitis, with photophobia. There were marked tachycardia and arterial hypertension.

The treatment consisted in the use of atropine, local application of a pomade containing cocaine, ultraviolet irradiation and general dietary measures. Recovery was extremely slow. The vasomotor phenomena persisted long after improvement had begun.

SCHLUTZ, Chicago.

GENESIS OF EXUDATIVE "ECZEMATOID." H. HULLSTRUNG, Monatschr. f. Kinderh. **80**:1, 1939.

This is an unnecessarily verbose article, taking 39 pages to record some observations on the cutaneous manifestations of exudative diathesis.

The writer first discusses exudative diathesis and the theories as to its cause. By "eczematoid" he means the various cutaneous manifestations that develop at any time thrŏugh life. The object of the study is to determine the sugar tolerance of the patient. He concludes that hypoglycemia is frequently present. Likewise the dextrose tolerance test in such persons gives abnormal curves, and the injection of epinephrine hydrochloride leads to no increase in blood sugar. He then goes into a philosophic analysis of the relation of these findings to abnormalities in internal secretion and concludes, by rather complicated reasoning, that in these persons there is an underfunction of the hypophysis, adrenals and pancreas and of the glycogen-building apparatus of the liver. This disturbance of internal secretion is congenital. The allergy of such patients is probably incidental.

GERSTLEY, Chicago.

PROBLEMS OF SO-CALLED INFANTILE ECZEMA. ATOPIC DERMATITIS. P. TACHAU, Acta dermat.-venereol. **20**:42 (Feb.) 1939.

The term "atopic dermatitis" is discussed. More than 50 per cent of the children with atopic dermatitis whom the author studied had a family or personal history of allergy. Scratch and intradermal tests and passive transfer of Prausnitz-Küstner reagins to different foods and inhalants gave positive results in almost all the cases. The author believes that the clinical facts point toward sensitization to inhalants as the etiologic factor in a considerable number of cases.

Atopic dermatitis in infancy is characterized by delayed lichenification and a tendency toward secondary eczematization. Common sites of predilection, besides the antecubital and popliteal spaces and the inguinal folds, are the face and the scalp.

Although itching is one of the outstanding features of atopic dermatitis, it was lacking in a few of the author's cases. Serious nervous disturbances are not found in infants with atopic dermatitis.

ASTRACHAN, New York. [ARCH. DERMAT. & SYPH.]

PURPURA SOLARIS. C. BERLIN, Acta dermat.-venereol. **20**:77 (Feb.) 1939.

The author describes a new form of purpura, the origin of which he attributes to changes in the skin caused by the sun's rays. Twenty-five cases were observed. The condition occurs more frequently in old than in young persons and is characterized by hemorrhagic spots of various sizes and shapes. The eruption is confined to the uncovered parts of the body and is always seen on the backs of the hands. The region of the skin on which the purpura appears presents changes which can be attributed to the influence of the sun, namely, scaling, some telangiectasia, dryness, atrophy and freckling. The condition does not cause any subjective symptoms and disappears completely after five to ten days, without leaving pigmentation.

A specific symptom, the nail pressure phenomenon, is described. A detailed description of 4 cases with histologic and other laboratory data is given.

A table summarizing all 25 cases is attached.

ASTRACHAN, New York. [ARCH. DERMAT. & SYPH.]

MULTIPLE ABSCESSES OF THE SWEAT GLANDS IN A BABY. H. B. VAN HAREN, Maandschr. v. kindergeneesk. **8**:355 (June) 1939.

A baby 17 days old had periporitides and multiple abscesses of the sweat glands, caused by Staphylococcus pyogenes aureus; a histologic examination was made. There was quick recovery after application of an unguent of sulfur and amorphous calcium carbonate. A short survey of the literature is included.

VAN CREVELD, Amsterdam, Netherlands.

Surgery and Orthopedics

CIRCUMCISION: HISTORICAL REVIEW. P. WRANA, Arch. Pediat. **56**:385 (June) 1939.

This is an excellent historical review of one of the oldest operations recorded. The custom of circumcision existed before 2,400 B. C.

ORR, Buffalo.

PHYSICAL THERAPY IN ORTHOPEDICS. K. HULDSCHINSKY, Brit. J. Phys. Med. **2**:124 (May) 1939.

Physical therapy methods used today in orthopedic practice comprise the whole of the fields of physical mechanics, optics, thermostatics and electricity; even acoustics is employed as an aid to the rhythmic movements of orthopedic gymnastics, and it helps to raise the muscle tone through the influence of the mind. Radiation treatment has almost completely ousted surgical procedures for tuberculosis of bones and joints. Electrical treatment is used for nerve paralysis, especially of the infantile form. Its only purpose is to protect the muscle from atrophy till the nerve lesion heals. The efficacy of roentgen irradiation of parts of the spine afflicted with poliomyelitis is not clear, nor is that of roentgen treatment for tuberculous joints.

HARTMAN, Cleveland.

CARE AND CURE OF THE CRIPPLED CHILD IN THE SOUTH-EAST OF SCOTLAND. W. A. COCHRANE, Edinburgh M. J. **46**:613 (Oct.) 1939.

This communication comes from the Princess Margaret Rose Hospital, Edinburgh, and the associated clinics. The writer discusses the early detection and

outpatient treatment of children with crippling defects. These disablements are enumerated. Early recognition and prompt treatment are necessary if the best results are to be obtained.

By means of 35 clinics, the hospital undertakes the responsibility for more than 1,400 outpatients in the aforementioned Scottish region. The type of organization which has been developed is emphasized. The surgical treatment required is highly specialized, but it is just as important that the organization be properly developed. The hospital building and the orthopedic setup are described as well as the social side of the scheme and the vocational training of the crippled. The plan contemplates the prevention as well as the cure of crippling.

New legislation will have to be provided for dealing with adults who have suffered from juvenile crippling, for organized orthopedic services for adults are incomplete in Scotland.

NEFF, Kansas City, Mo.

CYSTIC HYGROMA OF THE NECK. A. G. H. CLAY, Proc. Roy. Soc. Med. **32**:1379 (Sept.) 1939.

Cystic hygroma of the neck is reported in a boy 8 weeks old. Four days after birth a lump on the right side of the neck was noticed. It did not increase in size. On admission to the hospital the infant had a soft superficial swelling on the right side of the neck above the clavicle. This swelling was scarcely palpable until the child cried; then it became tense and fluctuant and about the size of a walnut. Although covered by normal skin, which was freely movable over it, the swelling was fixed deeply. On aspiration a clear sterile serous fluid was withdrawn. A roentgenogram of the chest showed an enlarged thymus.

WILLIAMSON, New Orleans.

TREATMENT OF SEVERE BURNS WITH ADRENAL CORTEX AND VITAMIN C. E. EINHAUSER, München. med. Wchnschr. **86**:444 (March 24) 1939.

In severe burns the intoxication with products of protein decomposition is important. There is injury to the capillary walls, permitting serum to pass from the blood to the interstitial spaces. Impairment of the exchange of metabolites between the blood and the tissues occurs, which injures the tissues, including the adrenal cortex. The author demonstrated the efficacy of adrenal cortex and ascorbic acid in the treatment of burns in guinea pigs. Subsequently several children with severe burns were treated similarly. Einhauser reports in detail the case of a 13 year old boy with a second and a third degree burn involving 55 per cent and 30 per cent, respectively, of the body surface. In addition to the usual treatment for burns, the boy received five daily injections of an adrenal cortex preparation equivalent to 20 Gm. of the fresh gland and two daily intravenous injections of ascorbic acid, totaling 1,500 mg., for three weeks. Frequent transfusions were given over a period of five weeks. The early course was stormy, but after ten months the boy's progress is encouraging. The author feels that his results warrant further trial of this form of treatment.

BRAHDY, Mount Vernon, N. Y.

THE INTEREST OF THE PEDIATRICIAN IN THE TREATMENT AND PREVENTION OF ORTHOPEDIC DISEASES. L. STOLTENBERG, Acta pædiat. **24**:320, 1939.

A large number of children exhibit seriously defective posture as well as flatfoot. Both disorders are apparently on the increase, possibly in relation to the acceleration in rates of growth and development that has taken place in civilized countries during the last two decades. Although the treatment of kyphosis, scoliosis, lordosis and flatfoot is usually delegated to the orthopedic surgeon, it is the duty of the pediatrician to take steps to prevent their occurrence.

MCCUNE, New York.

Teeth and Dentistry

ANTHROPOLOGIC ASPECTS OF DENTOFACIAL DEFORMITIES. HARRY BERNARD WRIGHT, Am. J. Orthodontics **25**:218 (March) 1939.

Deformities of the dentofacial structures are only one manifestation of growth disturbances affecting the entire organism. Factors causing such deformities were just as active in primitive as in modern man. Todd, Hellmann, Brodie and others are cited with respect to the relation of function and the form of bone.

LEWIS, Dayton. [ANGLE ORTHODONTIST]

* STUDIES ON DENTAL CARIES: IV. TOOTH MORTALITY IN ELEMENTARY SCHOOL CHILDREN. J. W. KNUTSON and H. KLEIN, Pub. Health Rep. **53**:1021, 1938.

Measures for dealing with the problem are discussed, and it is concluded that examination of the lower right (or left) first molars affords a reliable method for the comparison of "tooth mortality rates" in children. (NAR, 8:504.)

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* STUDIES ON DENTAL CARIES: VII. SEX DIFFERENCES IN DENTAL CARIES EXPERIENCE OF ELEMENTARY SCHOOL CHILDREN. H. KLEIN and C. E. PALMER, Pub. Health Rep. **53**:1685, 1938.

Results derived from an analysis of dental examinations of 2,232 boys and 2,184 girls indicate that the higher incidence of caries in girls as compared with boys of the same chronologic age is explained quantitatively by the fact that girls, because their teeth erupt earlier than do those of boys, are exposed longer to the risk of attack by caries than are boys. On the basis of these findings, the conclusion is reached that girls show no greater susceptibilty to attack by dental caries than boys. (Authors' summary.)

* Reprinted by courtesy of *Child Development Abstracts and Bibliography.* [ANGLE ORTHODONTIST]

RELATION OF VITAMIN D AND MINERAL DEFICIENCIES TO DENTAL CARIES. G. F. TAYLOR and C. D. M. DAY, Brit. M. J. **1**:919 (May 6) 1939.

The authors studied a group of children in the northern part of India in order to determine, if possible, the relation of diet, and particularly that of vitamin D, to the incidence of dental caries and marked rickets. In this group they found marked rickets general. They found general caries almost nil and a diet which was practically free from vitamin D, calcium and phosphorus. In another section of India, Lahore, a group of middle class children were studied, and they were on a so-called civilized diet, high in carbohydrates and sugar, and the incidence of caries in these children was much higher than in the children with rickets reported in this paper. The authors feel that "unrefined" foods and types of food which mechanically keep the teeth clean while being eaten have more effect than dietary balance.

ROYSTER, University, Va.

Miscellaneous

SALICYLATE POISONING. L. STANLEY SILL, Arch. Pediat. **56**:55 (Jan.) 1939.

A boy aged 8 years with Still's disease received 190 grains (12.2 Gm.) of sodium salicylate over a period of five days, after which he began to complain of abdominal pain. It was noticed that the patient was perspiring freely; the respirations were rapid; there was vomiting, and the child was apprehensive and had slight delirium.

Complete recovery in four days followed the use of dextrose solution intravenously and subcutaneously.

ORR, Buffalo.

THE CONSTITUTION OF THE ADOLESCENT CHILD. FRANZ SCHUCH, Arch. Pediat. **56**:199 (April) 1939.

This article deals with the physical constitution of the adolescent child and reports on work which is being done within the New York city school system. It is a survey of the work with its aims and its methods to improve the physical condition of the adolescent child.

ORR, Buffalo.

A CASE OF LEAD ENCEPHALOPATHY IN A BREAST-FED INFANT DUE TO THE USE OF LEAD NIPPLE SHIELDS BY THE MOTHER. MILTON RAPOPORT and ATHOL S. KENNEY, J. A. M. A. **112**:2040 (May 20) 1939.

The case of a 3½ month old infant is reported. The results of studies of the blood and of the spinal fluid are described.

HEERSMA, Kalamazoo, Mich.

AUTOLOGOUS AND HOMOLOGOUS TRANSFUSION OF HUMAN ASCITIC FLUID. H. A. DAVIS and J. F. BLALOCK JR., J. Clin. Investigation **18**:219 (March) 1939.

Evidence is presented to show that the transfusion of human ascitic fluid in group-compatible animals and human recipients is practicable. The authors suggest that the value of the fluid may be enhanced by concentration and state that the properties of the concentrated lyophile form of ascitic fluid are being investigated.

GUEST, Cincinnati.

THE EFFECT OF VITAMIN C ON LEAD POISONING. HARRY N. HOLMES, KATHRYN CAMPBELL and EDWARD J. AMBERG, J. Lab. & Clin. Med. **24**:1119 (Aug.) 1939.

There is a similarity between the symptoms of chronic lead poisoning and those of subclinical scurvy. In a series of cases of lead poisoning vitamin C was given in large doses. Within a week there was marked improvement. Vitamin C apparently combines with lead to form a complex compound which yields few free lead ions. This compound is absorbed by the liver, and then, through the bile, it is eliminated in the feces. The results of treatment with vitamin C were far superior to those of treatment with calcium.

McMURRAY, Galveston, Texas.

TREATMENT OF GASEOUS HYDROCYANIC ACID POISONING BY SODIUM THIOSULFATE AND SODIUM NITRITE COMBINATION. J. N. ETTELDORF, J. Pharmacol. & Exper. Therap. **66**:125 (June) 1939.

Etteldorf has previously shown that breathing hydrocyanic acid gas in concentrations below the convulsive level will not produce permanent poisoning and that the mechanism responsible for detoxification is not fatigued even by repeated daily exposures for many days. Exposure to the convulsive level for fifteen minutes is almost invariably fatal.

Because of the rapidity of the action and of the detoxification, it is difficult to determine whether a given animal would not recover without treatment. Dogs exposed to certain levels of hydrocyanic acid gas show salivation, tearing, defecation and urination, with increasing restlessness, always followed by convulsions. No deaths occurred in animals exposed for eight minutes to convulsive concentrations. From this point to the surely fatal exposure of fifteen minutes the results were variable with or without the sodium thiosulfate–sodium nitrite combination by vein. It seems then that this remedy is not certain to be of value unless the otherwise fatal exposure to hydrocyanic acid gas has not been reached.

PILCHER, Cleveland.

SULFANILAMIDE THERAPY FOR CHILDREN. JEROME L. KOHN, M. Clin. North America **23**:697 (May) 1939.

This paper is based on an experience in the treatment of 125 children with the drug. The usual dose was somewhat greater than that generally recommended

for adults, i. e., 0.2 Gm. kilogram (0.1 Gm. per pound) of body weight in twenty-four hours. Sodium bicarbonate was given only when alkalinity of the urine was desired. The efficacy of sulfanilamide in the treatment of hemolytic streptoccccic infections is well established. For infections involving bony structures its effectiveness is greatly reduced. Metastatic involvement of bone may develop in the face of otherwise successful treatment. The drug is of great value for streptococcic infections of the pleura and of the peritoneum, although it may become necessary to drain the pleural cavity and (occasionally) localized peritoneal abscesses.

Untoward effects of the drug should always be anticipated. Cyanosis not due to destruction of blood may be disregarded. Idiosyncrasy to sulfanilamide occurs less frequently in children than in adults. It usually manifests itself as acute hemolytic anemia. When such a condition appears the drug should be discontinued and the patient given a transfusion. In the more slowly developing form of anemia treatment need not be stopped, but small transfusions should be given when the value for hemoglobin reaches 50 per cent. Frequent examinations of the blood will disclose the appearance of leukopenia and agranulocytosis. If the reactions are mild, treatment may be continued if small transfusions are given. Maculopapular rashes may occur.

Treatment (under proper observation) may be continued for many weeks if necessary. For severe infections sulfanilamide should be continued for about a week after clinical cure, then given in gradually decreasing doses.

The combined use of immune serums and sulfanilamide in certain cases seems to increase the effectiveness of both agents.

EUGENE H. SMITH, Ogden, Utah.

CLINICAL EXPERIENCES WITH SULFANILAMIDE THERAPY (WITH SPECIAL REFERENCE TO TOXIC EFFECTS). REUBEN OTTENBERG, New York State J. Med. **39**:418 (March 1) 1939.

This excellent review from the Mount Sinai Hospital was read before the New York Academy of Medicine Oct. 6, 1938. The subject of sulfanilamide therapy is presented under various heads, and the toxic effects, including acute hemolytic anemia and agranulocytosis, are described in detail. The subject is well reviewed to that date.

AIKMAN, Rochester, N. Y.

MALIGNANCIES IN INFANCY AND CHILDHOOD. D. W. SCOTTI, New York State J. Med. **39**:1188 (June 15) 1939.

In a study of 64 malignant tumors occurring in infants and children Scotti gives an excellent review of the subject. The diagnosis in each instance was made by both gross and microscopic examination of the tissues. Seventy-two per cent of the tumors were encountered in children who were in the first five years of life and were about equally divided between boys and girls. Tumors of kidneys and adrenal glands led in order of frequency. Wilms's tumor was the most common. Although the death rate is high, malignant diseases should not be regarded as entirely hopeless. Such neoplasms as Wilms's tumor, endothelial myeloma and carcinoma of the thyroid are radiosensitive. Advances in the application of radiotherapy may reduce the high mortality rate.

AIKMAN, Rochester, N. Y.

SULFAPYRIDINE (DAGENAN) THERAPY. J. LEWIS BLANTON, West Virginia M. J. **35**:216 (May) 1939.

Thirty cases of infection recently treated with sulfapyridine are reported, 19 of pneumococcic and 11 of miscellaneous infections. Twenty of the patients were infants and children and 10 were adults.

Reference is made to published summaries of the literature, but the literature is not reviewed.

The dosage and methods were those established by various investigators. Complications and toxic symptoms are discussed. Brief histories of the cases are given. There were 2 deaths from pneumonia, both the patients being young infants with complications.

A. SHAWKEY, M.D., Charleston, W. Va.

IS IT EVER NECESSARY TO KILL A LIVE BABY? B. SOLOMONS, Brit. M. J. **1**:1175 (June 10) 1939.

After answering the question, "What is a live or viable fetus?" Solomons states that he deals "with the destruction of the fertilized ovum at any stage of the pregnancy." He does not refer to abortion under the supervision of competent and adequate consultation to save a mother's life. An abortion to relieve toxemia is discussed, together with renal toxemia and other complications of pregnancy. The discussion applies also to hemorrhage. The next topic discussed is cardiac disease, then severe exophthalmic goiter and like conditions—all of which, the author holds, justify abortion to a greater or lesser extent. Then he comes to the possible justification of the destruction of the fetus in the last three months of pregnancy, a period during which he considers the fetus to be probably viable. The various complications of phthisis are discussed, as well as the choice between cesarean section and the earlier interruptions of pregnancy. Uterine inertia is another phase discussed. The use of drugs for the induction of labor is discussed briefly and somewhat unsatisfactorily. The author really comes to no definite conclusions.

ROYSTER, University, Pa.

THE MODE OF ACTION OF DRUGS OF THE SULPHONAMIDE GROUP. JAMES MCINTOSH and LIONEL E. H. WHITBY, Lancet **1**:431 (Feb. 25) 1939.

The experiments reported support the view that drugs of the sulfonamide group have no stimulating action on leukocytes, phagocytic cells or the specific immune defenses of the body. Neither in vivo nor in vitro are they instantly active. There is a quantitative relation between the effective dose of the drug and the number of bacteria affected.

Drugs of the sulfonamide group are active on highly virulent organisms and those in the logarithmic phase of multiplication; they are inactive on "rough" organisms. These drugs are not simple germicides. They probably act by neutralization of some metabolic function or enzymatic activity.

LANGMANN, New York.

USE OF DERIVATIVES OF SULFANILAMIDE IN CHILDREN. G. PAISSEAU and R. VILLEY, Arch. de méd. d. enf. **42**:343 (June) 1939.

The brilliant results of this new type of chemotherapy are attested on every hand. Not only in frank invasions of the meningococcus, the streptococcus, the pneumococcus and the gonococcus but in obscure infections in which the microbial agent cannot be identified, various preparations of the sulfanilamide group are freely prescribed, in spite of various accidents and incidents attending their employment. A summary of results in carefully selected cases at the Trousseau Hospital for Children during the past few months is outlined. One of these concerned the successful treatment of an infant of 2 months suffering from meningococcic cerebrospinal meningitis. A further observation concerned a baby, also 2 months of age, stricken with streptococcic meningitis. In this instance cure was likewise complete and permanent. In a third case the use of sulfanilamide definitely checked the evolution of a grave complication of otitis media in a boy of 4½ years.

In contrast to these findings, the authors report failures in several cases of meningococcic meningitis and pneumococcic peritonitis in which the doses were carefully estimated; in other cases relapses occurred after apparent cure, in keeping with the experience of many other clinicians.

AMESSE, Denver.

SULFANILAMIDE THERAPEUTICS IN CHILDREN. J. BOISSERIE-LACROIX, Arch. de méd. d. enf. **42**:351 (June) 1939.

Conclusions from a questionnaire sent to representative pediatricians in Bordeaux and southwestern parts of France on the use of sulfanilamide in their practice are as follows:

1. If one excepts those suffering from hepatic disease, children, especially very young infants, tolerate these preparations with a minimum of danger.

2. Diseases in which remarkable results are commonly noted are: streptococcic invasions, adenoiditis, otitis, otomastoiditis and meningitis.

3. More doubtful results are seen in: otogenous meningitis, colon bacillus infections, gonococcic inflammations, bronchopneumonias, purulent pleurisies, scarlet fever and staphylococcic diseases.

4. Failures may be expected in purulent meningitis when the causative organism is other than the meningococcus.

AMESSE, Denver.

WHAT IS PREVENTIVE PEDIATRICS? VEEDER, Bull. Assoc. internat. de pédiat. prév. **5**:33, 1939.

Veeder sums up his interpretation of this subject in the following comprehensive paragraph:

Knowledge that will enable a child to be well-born; to come into the world strong and healthy; to thrive lustily and without halt during infancy; to develop and grow in a normal way during childhood; to be able physically and mentally to attain a sound education; to acquire good habits of living and avoid bad ones; to avoid psychological abnormalities so that he adjusts himself to his social environment; to pass through the difficult period of puberty and adolescence into a sound normal maturity; to prevent by scientific measures so far as we are able, the infectious diseases of childhood—these are some of the aims and objects of preventive pediatrics.

AMESSE, Denver.

"GIANNINA GASLINI" AT GENOA. M. BERGAMINI, Lattante **9**:511 (Dec.) 1938.

The institute "Giannina Gaslini" was opened in Genoa, Italy, on July 18, 1938. It is a children's hospital and institution consisting of seventeen buildings. These buildings include an infant asylumn, an orphanage, an outpatient department, a pediatric clinic, a surgical clinic and "contagious pavilions" for children with measles, diphtheria, whooping cough, scarlet fever and tuberculosis. Pictures of the various buildings are included in the descriptive article. HIGGINS, Boston.

ITALIAN CONTRIBUTIONS DURING 1938 TO THE STUDY OF INFANTS. F. LO PRESTI-SEMINERIO, Lattante **10**:251 (June) 1939.

During 1938, 750 pediatric articles were published in Italian, of which 350 dealt with children under 2 years of age. The subject matter of the articles on infants is reviewed in this seventeen page paper. HIGGINS, Boston.

PRESENT TENDENCIES OF GENERAL MEDICINE AND PEDIATRICS. WALTER PIAGGIO GARZON, Bol. Inst. internac. am. de protec. a la infancia **12**:499 (Jan.) 1939.

The author presents a panoramic view of medicine in relation to associated sciences, including social science. LESLIE, Evanston, Ill.

ACTION OF COUNTERIRRITANT MEDICATION IN THE NURSLING. D. TIMUS and V. PETRESCU-COMAN, Med. inf., Bucuresti **1**:217 (May-June) 1939.

The authors have studied factors of counterirritant application of cataplasms, baths and mustard packs. In this study they have counted the number of white cells and estimated the leukocytic formula for young infants one-half hour, one

hour, two hours and twenty-four hours after the application. They have noticed some modification in the number of white cells (up to 1,000 per cubic millimeter) but none of enough importance to enable one to speak of hyperleukocytosis. The polymorphonuclear neutrophils in general undergo no change.

The authors conclude that these applications do not produce modification in the form of leukocytosis or polymorphonuclear increase. Consequently, counter-irritant medications act by means of hyperemia and from the local cutaneous irritation and stimulate general circulation and secondarily pulmonary circulation.

FROM THE AUTHORS' SUMMARY.

THE HARMFULNESS OF WATER CHLORINATION AND ITS AVOIDANCE BY SILVERING ACCORDING TO THE CUMASINA METHOD. W. KRUSE, Arch. f. Hyg. **122**:177 (May) 1939.

Kruse emphasizes the advantages of disinfecting water, in swimming pools especially, by what he describes as an active silver and electric current method (the Cumasina method). It is of great disinfecting efficacy but entirely harmless at any concentration. It is without any unpleasant odor or taste. In speed of disinfection it is surpassed by chlorine only when the latter is in concentrations so high as to be injurious to the health of persons using the pool.

FALKENHEIM, Rochester, N. Y.

COMPARATIVE STUDIES OF THE ABSORPTION OF SODIUM SALICYLATE ADMINISTERED ORALLY AND RECTALLY. H. KRANZ, München. med. Wchnschr. **86**:1535 (Oct. 20) 1939.

Kranz studied the absorption of sodium salicylate following rectal and oral administration by giving the same amount of salt to the same patient by each route at different times and measuring the urinary excretion of the salt. After a single dose the total urinary excretion of salicylate was complete in forty-eight hours in 40 of 46 cases. For rectal administration the salt was dissolved in 100 cc. of water. The absorption was the same, whether the salt was given by mouth or by rectum. The average amount excreted in the urine was 87 per cent of the amount taken. The effect of fever was observed in 2 children. When afebrile, they excreted an average quantity after a trial dose of salicylate. Subsequently an acute febrile episode developed and the excretion of the salicylate in the urine decreased 30 per cent. This indicates splitting or destruction of the salicylate during fever.

BRAHDY, Mt. Vernon, N. Y.

AN INTERESTING CASE OF DIGITALIS POISONING. P. KISS and B. WOLLEK, Orvosi hetil. **83**:225 (March 11) 1939.

A 13 year old boy received 1.315 Gm. of digitalis and 1.27 Gm. of quinine in twenty-eight days for rheumatic pancarditis. The drugs were given seven days in succession, followed by a pause of two days. In the end tachycardia developed, and the well known symptoms of overdosage of digitalis (e. g., heart block) became evident only after a longer period of omission of the drug.

GÖTTCHE, Budapest, Hungary.

FIRST CHILDREN'S HOSPITAL IN SHANGHAI. Chinese M. J. **56**:177 (Aug.) 1939.

The new Shanghai Public Hospital for Children, on Moulmein Road, was opened July 12, 1939. It is an amalgamation of two institutions which had been functioning since the war began. There is room for 100 sick refugee children. The hospital has an outpatient department and a staff, with x-ray and operating rooms. The institution is appealing for further endowments.

NEFF, Kansas City, Mo.

ARTIFICAL FEVER THERAPY IN INFANTS AND CHILDREN BY INTRAVENOUS INJECTIONS OF TYPHOID-PARATYPHOID VACCINE AND PYRIFER. W. HEYMANN and L. S. ENRIGHT, Ann. pædiat. **154**:1 (Oct.) 1939.

Intravenous injections of typhoid-paratyphoid vaccine and of pyrifer (a nonspecific protein mixture prepared with extracts of fever-producing bacteria from nonpathogenic stacks) are recommended for pyretotherapy in infants and children. The routine procedure is described. It permits repetition of from four to six courses, each course consisting of three or four weeks of daily fever treatment, within a period of two or three years without harmful effects on patients of this age group.

Of 23 children with chorea, complete cure was accomplished in an average of seventeen days in 18. Artificial fever therapy had no influence on the occurrence of relapses. Rheumatic joints were treated with salicylates while fever therapy was being given, as salicylates have no effect on this type of artificially produced fever. Rheumatic carditis without heart failure was not a contraindication to pyretotherapy. Artificial fever therapy neither aggravated nor lessened the rheumatic state in 11 children.

Definite improvement was seen in 5 of 6 children with syphilitic interstitial keratitis, although no further benefit was obtained after the second week of treatment. It is therefore recommended that in cases of this kind artificial fever therapy be limited to daily treatments for two weeks.

Eleven children with syphilis of the central nervous system were given from four to six courses of fever therapy, each course consisting of three or four weeks of daily treatment. Eight of these patients were asymptomatic. Improvement in mental and speech disturbances, as well as in bladder symptoms, was seen in 1 child with dementia paralytica, 1 child with the tabetic form of dementia paralytica and 1 tabetic child. Pupillary symptoms, areflexia and complete optic atrophy did not improve. The Wassermann reaction of the spinal fluid became negative in 37 per cent of the 11 cases, improved quantitatively in 18 per cent and failed to show improvement in 45 per cent. The colloidal gold curves were reduced to negative in 17 per cent, were lowered in 50 per cent and remained unchanged in 33 per cent of the cases.

One encouraging result was obtained in a patient with encephalitis, most probably Schilder's disease.

Artificial fever therapy was of no value in cases of epilepsy, bronchial asthma, lipoid nephrosis and hereditary tremor. FROM AUTHORS' SUMMARY.

RESEARCHES ON A NEW GOLD PREPARATION, AURON B. A. M. ERNST, Nederl. tijdschr. v. geneesk. **83**:2047 (May 6) 1939.

The toxicity of two gold preparations, auron A and auron B, and the rapidity with which they were absorbed were examined in dogs and rabbits; 1.5 Gm. of auron A (soluble in water and in oil) administered under certain circumstances appeared to cause marked eosinophilia, while 5.5 Gm. of auron B (nonsoluble in water or oil) did not cause eosinophilia under the same circumstances. Roentgenologically it appeared that 50 mg. of auron A was absorbed within forty-eight hours, while the same quantity of auron B even after three months was clearly visible on roentgen examination. VAN CREVELD, Amsterdam, Netherlands.

POISONING BY OIL OF CHENOPODIUM. J. VAN LOOKEREN CAMPAGNE, Nederl. tijdschr. v. geneesk. **83**:5472 (Nov. 18) 1939.

In the province of Groningen, Netherlands, 4 children (15 and 19 months and 3 and 6 years old, respectively) have died of poisoning by oil of chenopodium within three years. None of them had been given the anthelmintic on medical prescription. VAN CREVELD, Amsterdam, Netherlands.

Society Transactions

CHICAGO PEDIATRIC SOCIETY

GERARD N. KROST, M.D., *President*

GEORGE F. MUNNS, M.D., *Secretary*

Regular Meeting, March 19, 1940

Relation of First Infection to Clinical Tuberculosis. DR. J. ARTHUR MYERS, Minneapolis.

The number of authentic cases of congenital tuberculosis reported in the world's literature is extremely small. Therefore, nearly all tuberculosis must be contracted after birth. Through the recognition of the contagiousness of this disease, almost 100,000 beds have been provided in sanatoriums and hospitals for tuberculous patients. The removal of these patients from their homes has prevented the spread of their tubercle bacilli in their communities. Modern aids in diagnosis are enabling physicians everywhere to detect disease in the lungs in a much earlier stage than in the past, so that many patients have their illness diagnosed before or soon after it becomes contagious. Modern methods of treatment, particularly collapse therapy, prevent the disease from becoming contagious in some patients and in others convert it to a noncontagious stage.

The protection of citizens against the bovine type of tubercle bacillus has been effected through inspection of meat, through pasteurization of dairy products and especially through the veterinarians' program of eradication of tuberculosis in cattle. The bovine type of tubercle bacillus is capable of causing all forms of tuberculosis in the human body. Therefore, the protection of man against tubercle bacilli of both the human and the bovine type has resulted in a marked decrease in the incidence of infection among children and young adults. In the midwestern part of this country, even in some of the cities, the infection attack rate is 1 per cent or less each year, both in children and in adults. Therefore, if this low rate is maintained, it is obvious that only 15 per cent of the children born at this time will be infected by the time they attain adulthood and only 60 per cent by the time they are 60 years old. Therefore, 45 per cent become infected for the first time in adult life. Obviously, it can no longer be said that nearly all tuberculous infection first occurs in childhood.

Another encouraging fact is that the infection attack rate apparently is decreasing, and indeed, already in some places the incidence of infection among children is extremely low. For example, Vadheim reports that a tuberculin-testing survey of all the children and teachers of Lincoln County, Minn., revealed only 5.2 per cent of reactors. Thus, the ideal situation may be closely approached in the next few decades, when nearly all children and adults escape tuberculous infection.

The first infection type of tuberculosis (primary complex) usually is an extremely benign disease. In fact, most persons, regardless of the age when it develops, are not aware of its presence unless they are tested with tuberculin. Among the children my associates and I have observed at Lymanhurst Health Center, we have not seen a single child who became seriously ill from the lesion of the primary tuberculosis complex itself. During the past decade, we have observed over 700 young adults, mostly students of nursing or of medicine, who became infected while in professional schools. In their bodies the primary tuberculosis complex was apparently as benign as it was in the bodies of the children under our observation.

With the small number of children and young adults infected with tubercle bacilli in certain parts of the country, there has been an opportunity to study the effects of first infection in infants, in children and in young adults, as well as the relation between this type and the reinfection, or clinical, type of tuberculosis. It is well known that not all persons in whom the primary tuberculosis complex (tuberculous infection) develops fall ill, and not all who fall ill die of tuberculosis. However, evidence is accruing to lead us to believe that at some time in life in 25 per cent or more of infected persons the clinical type of tuberculosis develops somewhere in their bodies.

We have also studied the reinfection, or clinical, types of tuberculosis, which have developed in the bodies of persons who became infected, both as children and as adults. For example, among 1,622 infants from birth to 2 years of age, 209 reacted definitely to tuberculin and 65 questionably. In 10 of this group an acute reinfection form of tuberculosis, such as meningitis, pneumonia or miliary disease, developed; all died. In no case was there any evidence that the first infection type of disease in itself caused any destruction. However, this type of disease did sensitize the tissues and endogenously cause the reinfections. Among 2,706 children 3 to 5 years of age, inclusive, 500 were definite reactors to tuberculin, and 130 presented questionable reactions. In none of these infected children did any reinfection type of tuberculosis, acute or chronic, develop during the period of childhood.

In another study we reported on a group of 446 children who reacted to the tuberculin test but were apparently healthy. When they were traced over a period of ten or more years it was found that in 15 per cent clinical forms of tuberculosis had developed. The average age at which the clinical disease was detected was approximately 18 years.

Among 441 students of nursing and medicine who reacted to tuberculin when admitted to their respective schools, we have seen clinical tuberculosis develop in 15. In a group of 643 young adults who became infected while under our observation, we have seen 10 in whom later only pleurisy with effusion developed and 32 in whom lesions subsequently developed which we believe are of the clinical type. A few of these had tuberculous pneumonia, but none had meningitis or miliary disease. In no case did we see clinical tuberculosis develop in a person who did not react to tuberculin.

We are of the opinion that those who have been infected with tubercle bacilli may become reinfected exogenously through exposure to persons with contagious tuberculosis, but we do not believe this occurs frequently in places like the midwestern part of this country, where only 1 per cent of the population becomes infected each year. It is our opinion that the majority of the reinfections which produce clinical disease are endogenous in origin, that is, the bacilli are derived from the lesions of the primary complexes. If this is true, it is extremely discouraging, as once the primary tuberculosis complex is allowed to develop the medical profession has no drug and no mechanical or other procedure which will disinfect the body of tubercle bacilli. Therefore, the only true solution of the problem is to prevent the body from becoming infected with tubercle bacilli. This is well within the realm of physical possibility and is being carried out with a great degree of success in many parts of this country.

DISCUSSION

DR. JOSEPH GREENGARD: I consider it a privilege to be called on to discuss this paper. Dr. Myers' contribution to this subject has been important and most enlightening. The subject is a significant one, and complete agreement of all investigators with the views expressed is certainly lacking. There are many who feel that a primary tuberculous infection may contribute to an increase in resistance to subsequent infection and so may not be altogether a bad thing. This basic concept is therefore a controversial one. It seems to me logical, however, to draw

the conclusions the essayist seems to indicate—that the primary infection, though in itself benign, is the essential basis for the development of reinfection types of tuberculosis. In other words, chronic progressive pulmonary tuberculosis of the adult, for example, cannot occur unless the person has first experienced a primary infection. Consequently, if primary tuberculosis can be prevented, a great step has been taken in the control of this serious disease. If this is true, one may then question the logic of procedures devised with the purpose of producing an active immunity to tuberculosis. I am referring to the method recommended by Calmette and his followers. I know there is great difference of opinion as to the degree of immunity or resistance to tuberculosis developed in natural infections. I should like to hear Dr. Myers discuss this.

Physicians who are practicing pediatrics deal principally with the primary infection types of tuberculosis. Occurrence of the adult type in children is relatively rare, although there are certain institutions, such as the Cook County Hospital, where one sees a good deal of it. Primary tuberculosis may be said to be a benign type in general, although such a statement must be qualified by two facts. In the first place, in young children with the primary type there is a definite tendency for violent exudative phenomena, which may be dangerous to develop. The second factor is the great tendency for wide dissemination of the infection during the evolution of primary tuberculosis. In point of time, generalization occurs during the first few months after infection. In young children, therefore, in whom the infection is recent, one must be careful in the clinical management, bearing this danger always uppermost in mind.

I have had the good fortune to observe a child at Cook County Hospital who illustrates beautifully the relation of the primary infection to clinical tuberculosis. This Mexican child entered the hospital at the age of 9 months, because her mother was found to have open pulmonary tuberculosis. She had therefore been subjected to intimate and long-continued exposure. On admission a massive primary infiltrate was demonstrated, involving the upper lobe of the left lung, and she also showed tuberculous osteoperiostitis of several of the small bones of the hand.

She was kept under observation for a little over two years, in which time the infiltrate gradually cleared and in clearing revealed a calcified parenchymal focus and secondarily infected glands, which were the source of the infiltrate. Her spina ventosa healed completely. She was discharged in good condition at the age of 30 months.

In 1938, at the age of about 8 years, she returned to the hospital and then showed a reinfection type of tuberculosis, with massive caseous pneumonia and a cavity on the right side. This was collapsed successfully, but soon involvement appeared on the other side.

This child furnishes a good illustration of the great tendency to healing with tuberculosis of a primary infection type, even when infection has been heavy and massive exudative lesions have occurred in a young child. When reinfection occurs, however, the situation is much graver.

DR. J. B. NOVAK: I have had no extensive experience in pediatrics, but my associates and I have had the opportunity of watching the reinfection type develop from the primary type of tuberculosis. We have not done such extensive work as Dr. Myers, but we have run across many questions that need answering.

We should like to know why the reinfection type does not develop in children until about the age of 12 years and why persons that have primary infection in adult life have the reinfection type within a year or two afterward. Why do some persons have reinfection types and others not?

In my opinion, most of this can be answered on the basis of either an endogenous or an exogenous reinfection. Both factors probably come into play. We know many reinfections develop from the endogenous factors, but that does not rule out the fact that patients can be reinfected from an exogenous focus.

We still exercise a tremendous amount of caution even though a person has had a primary infection. We caution these patients not to expose themselves to infection. We found, incidentally, that reinfection is always in direct proportion to the degree of intimacy of contact. The estimate that primary infection produces a reinfection type of tuberculosis in 30 per cent of the persons infected is rather high in my opinion. We found that the reinfection type develops in only 1 of 200 patients who have had transient contacts, while it appears in about 1 of 5 who have had intimate contact with a source of infection.

DR. J. W. MOORE, Danville, Ill.: I have a large number of children and young adults with first infections of tuberculosis under observation at the taxpayers' expense. I should like to know how often it is necessary to have them in for reexamination, with roentgen examinations and tuberculin tests, and to what age I should continue the observation. In other words, I want to know whether it should be continued to the age of 25, 30, or 35 or when I am justified in stopping it. I am interested in getting the most for the money—and it is taxpayers' money.

DR. JESSE GERSTLEY: May I ask Dr. Myers whether the tuberculin tests were made with the Mantoux or with the Pirquet technic? Sometimes the Mantoux test is positive and the Pirquet negative.

The conception that a positive tuberculin test signifies living tubercle bacilli is new to me. May I ask if this is now generally accepted? If so, would Dr. Myers describe the clinical or experimental work which has ruled out the idea I previously held, namely, that of tissue sensitivity?

DR. HENRY C. SWEANY: Nobody can hear Dr. Myers without being greatly interested, entertained and benefited by his presentation. I want to pay a tribute to him for the stand he has made in certain respects with regard to handling tuberculosis in children. Dr. Myers was one of the first to bring out emphatically that roentgen examination and not clinical methods was most important in discovering early tuberculosis in children. Tonight, he not only showed the advantage of roentgen examination but pointed out that even it has limitations.

I was interested in his report of primary tuberculosis in university students (700 I believe) in few of whom the severe disease developed from the primary lesions. This may give the impression that such infections are not serious. It must be remembered that infection is always far greater than morbidity, and the morbidity is greater than the mortality.

Dr. Myers discussed one group—the living; in our institution my associates and I have studied the other group—those who have died. We have performed autopsies on approximately 1,300 patients, 75 per cent of whom we felt died as a result of an infection dating back to the primary infection. On comparison of these two sets of figures one might think there was a discrepancy that could not be explained. However, our autopsies represent fatal infections among over 3,000,000 people from all walks of life. Dr. Myers' figures give the number of infected persons among university students in the state of Minnesota; they have not died, and only a few have even had the severe form of the disease. Our patients were from a cross section of the population of a large city who have died, and those of Dr. Myers were from the student body of a large university, who have been watched and cared for and in whom the disease has not been permitted to develop.

Why does the disease develop in certain patients? Why is it that certain patients are infected and the disease then runs only a brief benign course or lasts for a year and then heals, whereas in others a smoldering type of infection will develop that continues on and on and finally after several years progresses to terminal disease? Diet, number of bacilli and social status are factors, but it is the unpredictable nature of the disease that is important. One cannot tell yet in which patients a more advanced stage of the disease will develop and in which the infection will heal. That is why it is necessary to keep eternal vigilance.

It has been found that in the great majority of cases the local lesions become encapsulated and the bacilli die. I feel that the old ossified lesions may disseminate germs and have raised the question as to that possibility, but as a general rule these old lesions are sterile. Daughter colonies, however, may develop and cause the infection to persist. Primary lesions may become encapsulated and healed in 95 to 99 per cent of patients, but in the 1 patient in a hundred an overflowing lesion develops that will produce a daughter colony or an infiltrate or break through into a bronchus or blood vessel and produce disease later. Many times a bronchial lymph node will ultimately break into a bronchus and cause reinfection. At other times the bacilli may lie dormant in a slowly spreading process.

When a child is infected early, before the age of 5 years, and the disease does not develop at that time, yet he has an active lesion, the lesion may break down later, usually in young adult life. It seems to carry over the childhood age. But in the young adult the "lag," or latent period, is only two to five years. The young adult may have tuberculous pneumonia or other serious development, coming on rather quickly after the primary infection. Thus it may be seen that the disease process itself is focused in young adult life.

The question as to the cause of this has been raised. In the first place, there is a greater tendency to localization in the parenchyma in adults than in children. Also, the connective tissue does not develop as rapidly in adults as in children. There is in general a thinner capsule and a greater tendency to "overflow." Most of the lesions heal, as Dr. Myers has shown, but when they do break open they have a tendency to spread the disease more rapidly than in the child.

DR. WALTER H. WATTERSON, La Grange, Ill.: I always enjoy hearing Dr. Myers, and his expression of the conditions is so simply set forth that any one can understand. I think it was in 1938 that Watson, of London, brought out this theory—that the greater the reaction from the tuberculin test, the better the prognosis. I made some studies of that but am not ready to make a final report. May I have Dr. Myers' opinion of this?

DR. RALPH SPAETH: I wanted to ask some questions pertaining to group surveys of tuberculosis in children and in minors. In Chicago, under the relief administration, approximately 1,800 children are under the observation of the medical clinic conducted by the Children's and Minors' Service. These children and minors are examined for placement in foster homes. They are followed by periodic visits to the clinic. Routine tuberculin tests, among others, are performed both before and at various intervals after placement in foster homes. Certain problems arise in connection with this arrangement. They are specifically as follows:

1. What danger is there in doing routine tuberculin tests on children who have had these tests (with positive or negative results) previously? In the past two weeks, of approximately 50 subjects given tuberculin tests 2 or 3 had marked local and constitutional reactions. One child had a swelling which extended to the elbow, and another had a temperature of 102 F., with malaise and a suggestion of rigidity of the neck. Uneventful recovery took place after several days. About a year ago, a child had a cough, malaise and fever of over a week's duration after a tuberculin test. I wonder how much danger there is in repeating tuberculin tests on children who might pass from clinic to clinic. In these tests, my associates and I use a solution intermediate between the first and second strengths of purified protein derivative as commercially available. The solution (0.1 cc.) is given intracutaneously.

2. How feasible is the Vollmer test, and to what extent can it replace the others?

3. If we use the Mautoux test, what dilution and what material should be used so that the possibility of reactions is minimal and the detection of tuberculin sensitivity normal?

4. When children who are to be placed in foster homes have positive tuberculin tests, either alone or in combination with evidences of healed childhood tuberculosis as revealed by roentgen examination, how often should the roentgen examinations be reported?

We are particularly interested in this phase of the study of tuberculosis, because it involves a financial and administrative problem.

DR. ARTHUR J. MYERS: Immunity was not mentioned in my presentation, but since the subject has been raised I shall discuss it briefly. Induction of artificial immunity was attempted after it was known that an attack of certain diseases confers a definite immunity, and therefore there was an excellent reason for hoping to develop a method of artificial immunization against such diseases. As one attack of tuberculosis in an animal or a human being apparently does not confer a satisfactory immunity, it would appear that any attempt to produce immunity artificially starts with a shaky premise.

Since 1885 there has been a tremendous amount of work done to develop an artificial immunity in animals and also in man. I think perhaps the best lesson comes from the veterinarians, who have employed various methods in thousands of animals in all parts of the world. There was a wave of enthusiasm between 1901 and 1905 for von Behring's bovovaccine. Many felt that tuberculosis would soon be of only historical interest. Then it was found that this substance did not produce immunity. Calmette's vaccine (BCG) does not differ much from von Behring's except that von Behring used the human type and Calmette the bovine type of tubercle bacillus. BCG has been used extensively in the hope that it would solve the problem of tuberculosis among cattle. In North America it has been practically discarded, because it was found that the vaccine does not kill the virulent tubercle bacilli already present in the body nor does it prevent reinfection from developing in the animal. Moreover, it was found that among the animals in which immunization had been attempted, as many and as significant lesions developed as among the controls.

Why reinfection does not develop until the age of 12 years was told, I think, by Dr. Sweany; also why reinfection develops in some persons and not in others. It has been known since 1885 that exogenous reinfection can be produced in animals, and there is good evidence that many persons who have had a primary infection and remain in direct contact with persons with contagious tuberculosis fall ill with clinical tuberculosis.

The discouraging fact is that endogenous reinfections occur from the lesions of the primary complex. It is discouraging, because when primary complexes develop there is so little one can do to prevent endogenous reinfection from them. Tuberculosis is different from syphilis in that drugs are available to disinfect the body of spirochetes, whereas there is available no drug or mechanical procedure which destroys tubercle bacilli in the body.

The question has been asked how often to reexamine and at what age. We have not found it practical to make a roentgen reexamination of children who react to tuberculin until the approach of adolescence, but thereafter we think it important that annual examinations be made as long as the person reacts to tuberculin. Dr. Sweany pointed out that in many persons the tubercle bacilli die out completely, and there is good evidence that in those in whom the bacilli die the sensitiveness to tuberculin disappears. Therefore, the fact that a person reacts to tuberculin does not mean that he will always do so. We have adopted the policy of repeating the tuberculin test every three to five years.

It has been expensive to repeat roentgen examinations, but with the new rapid methods of making films the cost has been so reduced that there will be practically no reason why examinations should not be repeated with considerable frequency in the future. Until physicians can make roentgen films of all patients who come to their offices or institutions as frequently as they feel they should be made, accomplishments will be limited. Roentgen examination of the chest should be routine, just as urinalysis is at present.

Most of the early tuberculin tests made by my associates and me were made by the dermal method of Pirquet, but for approximately ten years we have used the intracutaneous method of Mantoux. We have seen no reason to change to one of the other methods of administration, such as the tape or patch method. These are too time consuming when compared with the intracutaneous method.

The question as to the degree of reaction and prognosis has been discussed pro and con, but I do not know of any extensive or significant work that shows any relation between the intensity of the reaction and either the present or the future extension of the disease.

The best lesson concerning tuberculosis is from the veterinarians of this country. They went through the entire gamut of diagnosis, treatment and prevention and finally came to one point—that every animal that has a reaction to the tuberculin test has foci of living tubercle bacilli in its body. And since there was no way to tell which would later become ill, the only solution was to treat them all alike. Veterinarians have administered more than two hundred and seventeen million tests to cattle in this country since 1917. They have slaughtered and made postmortem examinations of more than 3,700,000 reactors. The tuberculin test has proved so specific that the disease is rapidly approaching eradication throughout the entire nation.

For tuberculosis of human beings there is the same specific tuberculin test that the veterinarians use. Once one finds a tuberculous patient one has, or should have, a place in which to isolate him if his disease is contagious. To remove a contagious patient from his home to a sanatorium is just as effective from the standpoint of the health of the community as is slaughter for the health of the cattle herds.

With collapse therapy, the disease often can be converted from the contagious to the noncontagious stage. Thus, where the veterinarians slaughter, physicians isolate, treat and educate, and by these methods there is no reason why they should not reduce tuberculosis to a minor disease within a reasonable period. The keynote must always be: Prevent infection.

Book Reviews

Virus and Rickettsial Diseases. Harvard School of Public Health Symposium Volume. Price, $6.50. Pp. 872, with 33 illustrations, 40 tables. Cambridge: Harvard University Press, 1940.

"Virus and Rickettsial Diseases" contains comprehensive reviews by twenty-two Harvard faculty members of significant information regarding the most important virus and rickettsial diseases. The concise presentation of material enables the reader to review the significant data quickly. Most chapters are completely outlined, and all have good bibliographies.

The volume is introduced with a chapter considering virus epidemiologic problems in general, the nature of viruses, their transmission, animal reservoirs, host-parasite relations and prevention of virus diseases. What is now a promising outlook for prevention is pictured as almost a certainty for the future if altered viruses can be so used as to permit an inapparent infection with permanent immunity but no ill effect. The discussion of the properties of viruses as they exist intracellularly in their host leads to a realization that further information must come from studies in intracellular biochemistry. A complete chapter on the insect vectors of virus diseases details the development of medical entomology, emphasizes how such scientific information has revolutionized understanding of the spread of many epidemic diseases and reveals the great complexity of natural reservoirs and methods of transmission.

Four chapters comprise a noteworthy accumulation of information devoted to variola, the use of smallpox vaccine virus, generalized vaccinia and neurologic complications of vaccination. It is emphasized that a constant high level of immunity in a community can be maintained only if vaccination is done regularly on all newcomers and that infancy is the time of choice for vaccination, since postvaccinal encephalitis is almost unknown at this period of life. Three chapters are devoted to the etiology, prevention and reporting of measles. Accumulating evidence indicates that the etiologic agent is most likely a virus and that convalescent serum or immune globulin if given in adequate dosage during the first five days after exposure usually protects, while if given between the fifth and ninth days it may have little effect or only slightly modify the course of the disease. Mumps is dealt with comprehensively from the standpoint of etiology, pathology, glandular and neurologic manifestations, treatment and prevention. The chapter dealing with the absorption of toxic and infectious material from various portions of the respiratory tract indicates the many possibilities in a most intriguing and promising field of investigation, which is likely to answer many questions pertaining to invasion by bacteria and viruses. Interesting data are already offered regarding the rapid invasion of the lymphatic system by a nonmotile agent such as the pneumococcus. The presentation regarding influenza reviews most of what is known in this field, especially the recent additions to knowledge regarding the virus etiology, and suggests that public health efforts at prevention may be premature until more specific etiologic information is available. Poliomyelitis is presented in three chapters, which deal especially with the epidemiology, clinical features, treatment and preventive measures. All phases are dealt with comprehensively. Three chapters are devoted to yellow fever, with a detailed consideration of the etiology, epidemiology and preparation and use of vaccine from modified tissue culture virus. Great numbers of people have been immunized recently, and 97 to 100 per cent show protective antibodies. Thus, in addition to smallpox, another virus disease is being successfully combated by antigen of tissue culture virus. Four chapters deal with a general survey of the rickettsial infections, as to diagnosis, classification, clinical features, epidemiology and immunity.

These diseases, recognized as the most complex of any of the infectious diseases of man, present problems of prevention and control which are most difficult because of the many vectors and mammalian reservoirs for each of the viruses. It is emphasized that the early attempts at prophylactic immunization with phenolized virus and later studies using formaldehydized virus have indicated that methods are available for almost complete prevention of this group of infections.

Altogether this volume is an outstanding example of the accumulation and presentation of valuable data regarding most of the important virus and rickettsial infections. It fills a need that has been felt for a long time.

Die Zwillingspathologie des Angeborenen Klumpfusses. By Dr. Karlheinz Idelberger. Beilageheft zur Zeitschrift für Orthopädie, volume 69. Price, paper, 8.60 marks. Pp. 95, with 2 illustrations and 27 tables. Stuttgart: Ferdinand Enke, 1939.

In the belief that ordinary methods of research into pathologic conditions in twins are incorrect and do not furnish reliable data, the author, accompanied by his wife, spent three years touring the Reich, exclusive of East Prussia, in an attempt to study the occurrence of clubfeet in twins. The completeness of the work was aided by the fact that for the last ten years in Germany every instance of congenital anomaly has been reported to the social service. There was, therefore, a total of 11,459 instances of clubfeet, of which official information was obtainable about 9,941 (86.8 per cent). One thousand five hundred and eighteen clubfooted persons (13.2 per cent) could not be traced. From twin births there were recorded instances in 304 persons, of whom 290, or 95.4 per cent, were examined, but data about only 242, or 83.4 per cent, were used in the compilation of this paper.

The investigation included both grandparents and the immediate family and showed, moreover, that a wrong diagnosis had been made in 48 instances, of which poliomyelitis was the condition actually present in 14 instances, spina bifida aperta in 6 and Little's disease in 5.

All persons were seen and personally examined, and they were traced through varied sources, in particular through clinics and the social service in rural communities. Only one third of the persons were from towns of over 5,000 inhabitants; so the incidence of clubfoot in them did not, consequently, represent the incidence given in hospital statistics. Only 1 instance of bilateral clubfoot in twins was found: in 28 year old women, 1 feebleminded. Of the series of 242 twins, 154 were boys (63.7 per cent) and 88 girls (36.3 per cent).

The theories and findings of the cause of clubfoot are discussed. The old theory of cramped space in the uterus is dismissed as a cause, as many normal fetuses are born through a narrowed birth canal (for instance, when tumors are present). The decubital ulcers on the ankles are believed not to be caused by the pressure of the uterine wall but to be the result, probably, of overstretched skin on prenatally deformed feet on which moderate pressure of the uterus causes decubitus.

The influence of inheritance is believed to be much stronger, although more difficult to prove, especially since Lange, in 1928, estimated that 75 to 80 per cent of clubfeet were due to intrauterine causes, mostly oligohydramnios. But clubfeet are found twice as often in boys as in girls; in 55 per cent of the cases the condition is bilateral and in 45 per cent unilateral, with involvement of the right and of the left foot equally common.

Idelberger found that congenital malformations were combined with clubfeet in 3.5 per cent of the cases, but that there are only a few instances of clubfoot in twins to be found in the literature.

If clubfoot were due to intrauterine causes, twins would be more affected because of narrower space. Therefore, one would expect to find more clubfooted persons among twins than there are among the average population, which is not the case, as is shown by this investigation. The percentage of occurrence of clubfeet in identical twins is much greater (30 per cent) than in dissimilar twins (1.7

per cent). Familial tendency is also shown by the fact that nearly every family with a child thus afflicted was found to have included some other person with the condition when its history was traced back two generations.

Pediatrics and Pediatric Nursing. By A. Graeme Mitchell, M.D., Echo K. Upham, R.N., and Elgie M. Wallinger, R.N. Price, $3.00. Pp. 575, with 83 illustrations. Philadelphia: W. B. Saunders Company, 1939.

The reader will find the arrangement of the material in this book convenient. The authors have divided it into such units as "Physical and Mental Growth and Development," "Nutrition," "Diseases of the Newborn" and "Diseases of Several Body Systems."

The many excellent illustrations, graphs and tables will undoubtedly benefit the pediatric student. The comments and summary at the end of each chapter help the reader to bear in mind the most important and outstanding points.

The chapter on "Mortality and Morbidity," followed by the one on "Prevention of Disease," seems significant and particularly valuable to the pediatric nurse.

Although the mental and emotional development of infants and children is discussed in the first unit, there is no interpretation of the facts, which have considerable significance in the nursing care of children. If instead of discussing "Theories of Behavior" and "Laws of Genetics" which really belong in other textbooks, the authors had substituted a chapter on the emotional problems of hospitalized children, such as adjusting to the hospital, homesickness, fear of treatments and the operating room, would not the book be of greater service to the student and the graduate nurse?

It would seem that a further explanation of play materials and interests of children at various ages would be desirable.

The reader will find disappointingly short the paragraphs on family life, school and convalescent homes. The nursery school teacher would question the function of the modern day nursery and nursery school as described in this book. Many psychologists would differ with the authors in regard to the treatment of thumb-sucking and masturbation, described in the chapter on "Functional Diseases of the Nervous System."

The unit on "Nutrition" is interesting, but a more detailed explanation of eating habits of children would be helpful.

The units on "Communicable Diseases" and "Diseases of Several Body Systems" are especially well written and organized. However, the subject of diseases of children seems greatly to overbalance the subject of nursing care. More discussion of the care of patients with cardiac disease and of various surgical and orthopedic conditions would aid the student.

As a whole, "Pediatrics and Pediatric Nursing" proves interesting, informative reading, but it is unfortunate that, while presenting to the nurse a fairly complete picture of diseases and routine procedures, it still leaves her with little understanding of the child himself and how best to guide his experiences in a hospital.

Handbook of Orthopaedic Surgery. By Alfred Rives Shands Jr., M.D., in collaboration with Richard Veverly Raney, M.D. Second edition. Price, $4.25. Pp. 567, with 154 illustrations. St. Louis: C. V. Mosby Company, 1940.

This is the second edition of Dr. Shands's excellent book on orthopedic surgery. To date there is no other book published which gives the medical student such a comprehensive picture of the subject. It is brief, yet adequate, and presented in a simple, understandable style.

While there have been some minor rearrangements, the text as a whole follows the same outline as his first edition. Many new illustrations have been added, and most of the previous illustrations have been redrawn. The subject matter and the carefully selected bibliography have been brought up to date.

The book is attractive; it is printed on thinner paper and is a handier size than the first volume.

Directory of Pediatric Societies*

INTERNATIONAL

International Association of Preventive Pediatrics

President: Prof. S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

International Congress of Pediatrics

American Committee:
Chairman: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
General Secretary: Dr. Kenneth D. Blackfan, 300 Longwood Ave., Boston.
Assistant Secretary-Treasurer: Dr. Charles F. McKhann, 300 Longwood Ave., Boston.

Canadian Committee:
Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.
Place: Boston, Mass. Time: Postponed indefinitely.

International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

FOREIGN

Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

British Paediatric Association

President: Prof. Charles McNeil, 44 Heriot Row, Edinburgh, Scotland.
Secretary: Dr. Arthur G. Maitland Jones, 31A Weymouth St., London, W. 1.

Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. E. Gjørup, Dronning Louises Børnehospital, Copenhagen.

Paediatrický Spolok na Slovensku

President: Dr. A. J. C. Chura, Lazaretská 11, Bratislava.
Secretary: Dr. P. Rados, Lazaretská 6, Bratislava.
Place: Pediatric Clinic, University Bratislava. Time: Six times a year.

Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. E. A. Cockayne, 98 Harley St., London, W. 1, England.
Secretary: Dr. R. Lightwood, 86 Brook St., London, W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

PALESTINE JEWISH MEDICAL ASSOCIATION, SECTION OF PHYSICIANS OF CHILDREN'S DISEASES

President: Prof. S. Rosenbaum, 26 Bialikstr., Tel Aviv.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

SOCIEDAD CUBANA DE PEDIATRÍA

President: Dr. Clemente Inclan Costa, Manrique 48, Habana.
Secretary: Dr. Serafin Falcon Lopez, Calle B No. 356, Vedado, Habana.
Place: Cátedra de Clinica Infantil, Hospital Mercedes, Habana. Time: Last Wednesday of every month.

SOCIEDAD MEXICANA DE PEDIATRIA

President: Dr. Jorge Muñoz Turnbull, Av. Alvaro Obregón 186, Mexico, D. F.
Secretary: Dr. Fernando López Clares, Medellín 191, Mexico, D. F.

SOCIÉTÉ DE PÉDIATRIE DE PARIS

President: Dr. B. Weill-Hallé, 49 Avenue Raymond Poincaré, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue du Pré aux Clercs, Paris, France.
Place: Hôpital des Enfants Malades, 49 Rue de Sèvres. Time: 4:30 p. m., third Thursday of every month.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON PEDIATRICS

Chairman: Dr. Julias H. Hess, Chicago.
Secretary: Dr. Hugh L. Dwyer, 315 Alameda Rd., Kansas City, Mo.
Place: Cleveland.

AMERICAN ACADEMY OF PEDIATRICS

President: Dr. J. B. Bilderback, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.
Place: Hotel Peabody, Memphis, Tenn. Time: Nov. 18-20, 1940.
Region I. Place: New Haven, Conn. Time: March 27-29, 1941.
Region II. Place: Richmond, Va.
Region III. Place: Palmer House, Chicago.
Region IV. Place. San Francisco.

AMERICAN HOSPITAL ASSOCIATION, CHILDREN'S HOSPITAL SECTION

Chairman: Miss Mabel Binner, Children's Memorial Hospital, Chicago.
Secretary: Winifred Culbertson, Children's Convalescent Hospital, Cincinnati.

AMERICAN PEDIATRIC SOCIETY

President: Dr. Thomas B. Cooley, 1728 Seminole Ave., Detroit.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis.

CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. L. M. Lindsay, 815 Medical Arts Bldg., Montreal.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, Hospital for Sick Children, Toronto.

Society for Pediatric Research

President: Dr. A. Ashley Weech, 3975 Broadway, New York.
Secretary: Dr. Mitchell I. Rubin, 1740 Bainbridge St., Philadelphia.

SECTIONAL

Intermountain Pediatric Society

President: Dr. Eugene Smith, 385–24th St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

New England Pediatric Society

President: Dr. R. Cannon Eley, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. James Marvin Baty, 319 Longwood Ave., Boston.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

North Pacific Pediatric Society

President: Dr. S. G. Henricke, 4215 N. E. Hancock St., Portland, Ore.
Secretary: Dr. J. S. Backstrand, 388 State St., Salem, Ore.

Rocky Mountain Pediatric Society

President: Dr. John Schoonover, Republic Bldg., Denver.
Secretary: Dr. F. Craig Johnson, 1850 Gilpin St., Denver.
Place: Children's Hospital, Denver. Time: First Saturday of each month from September to May, inclusive, 7:30 p. m.

Southern Medical Association, Section of Pediatrics

Chairman: Dr. Hughes Kennedy Jr., Highland Plaza Apts., Birmingham, Ala.
Secretary: Dr. Warren W. Quillian, 227 Aragon Ave., Coral Gables, Fla.

STATE

Alabama Pediatric Society

President: Dr. Stewart H. Welch, 1117 S. 20th St., Birmingham.
Secretary-Treasurer: Dr. William R. Britton, 4 Catoma St., Montgomery.

Arkansas State Pediatric Association

Chairman: Dr. G. D. Murphy, 112 Peach St., El Dorado.
Secretary: Dr. Madeline M. Melson, 909 Main St., Little Rock.

California State Medical Society, Section on Pediatrics

Chairman: Dr. Harold K. Faber, 2341 Clay St., San Francisco.
Secretary: Dr. Hartzel H. Ray, 23–2d Ave., San Mateo.

Florida State Pediatric Society

President: Dr. Warren W. Quillian, 227 Aragon Ave., Coral Gables.
Secretary: Dr. George N. Leonard, 605 Lincoln Rd., Miami Beach.
Place: Concurrent with state association meeting at time of convention.

Georgia Pediatric Society

President: Dr. L. H. Muse, 384 Peachtree St. N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.

Hezekiah Beardsley Pediatric Club of Connecticut

President: Dr. Oliver L. Stringfield, 1416 Bedford St., Stamford.
Secretary: Dr. Joseph L. Hetzel, 103 N. Main St., Waterbury.
Time: Three meetings a year.

Illinois State Medical Society, Section on Pediatrics

Chairman: Dr. H. William Elghammer, 5307 Hyde Park Blvd., Chicago.
Secretary: Dr. Bert I. Beverly, 8 S. Michigan Ave., Chicago.
Place: Peoria. Time: May 22, 1940.

Indiana State Pediatric Society

President: Dr. O. N. Torian, 23 E. Ohio St., Indianapolis.
Secretary-Treasurer: Dr. Mathew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

Iowa Pediatric Society

President: Dr. Morgan J. Foster, 837 Higley Bldg., Cedar Rapids.
Secretary-Treasurer: Dr. Arnold M. Smythe, 406–6th Ave., Des Moines.

Louisiana State Pediatric Society

President: Dr. H. Aubrey White, Alexandria.
Secretary-Treasurer: Dr. W. C. Rivenbark, 220 Physicians and Surgeons Bldg., New Orleans.

Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. Paul W. Beaven, 26 S. Goodman St., Rochester.
Secretary: Dr. Leslie O. Ashton, 850 Park Ave., New York.

Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. John M. Higgins, 600 S. Wilbur Ave., Sayre.
Secretary: Dr. John D. Sturgeon Jr., 22 N. Gallatin Ave., Uniontown.

Michigan State Medical Society, Pediatric Section

Chairman: Dr. Allan L. Richardson, 3001 W. Grand Blvd., Detroit.
Secretary: Dr. Ward L. Chadwick, 524 Medical Arts Bldg., Grand Rapids.

Mississippi State Pediatric Society

President: Dr. Guy C. Jarratt, 920 Crawford St., Vicksburg.
Secretary-Treasurer: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Place: To be designated by society. Time: Yearly.

Nebraska Pediatric Society

President: Dr. Herman M. Jahr, 1120 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. Elmer W. Bantin, 440 Aquila Court Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

North Carolina Pediatric Society

President: Dr. Marion Y. Keith, 369 N. Elm St., Greensboro.
Secretary: Dr. G. W. Kutscher Jr., 176 Woodfin St., Asheville.

Oklahoma State Pediatric Society

President: Dr. J. B. Snow, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 1200 N. Walker St., Oklahoma City.
Place: Oklahoma Club. Time: 6:30 p. m., fourth Friday of each alternate month from September to May, inclusive.

South Carolina Pediatric Society

President: Dr. D. Lesesne Smith Jr., 187 Oakland Ave., Spartanburg.
Secretary-Treasurer: Dr. William Weston Jr., 1428 Lady St., Columbia.

Tennessee State Pediatric Society

President: Dr. William E. Van Order, 546 McCallie Ave., Chattanooga.
Secretary: Dr. Kinsey M. Buck, 1193 Madison Ave., Memphis.

Texas Pediatric Society

President: Dr. T. A. Tumbleson, 2100 North St., Beaumont.
Secretary-Treasurer: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Place: Dallas. Time: May 15, 1940.

Virginia Pediatric Society

President: Dr. W. Ambrose McGee, 616 W. Grace St., Richmond.
Secretary: Dr. John M. Bishop, Medical Arts Bldg., Roanoke.

West Virginia State Medical Society, Section on Pediatrics

President: Dr. J. L. Blanton, 207 Fairmont Ave., Fairmont.
Secretary: Dr. Theresa O. Snaith, 450 Center Ave., Weston.

LOCAL

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. N. C. Wetzel, 2103 Adelbert Rd., Cleveland.
Secretary: Dr. Fred Rittenger, 10515 Carnegie Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. T. B. Verner, 170 St. George St., Toronto, Canada.
Secretary: Dr. D. V. Hutton, 170 St. George St., Toronto, Canada.

Bronx Pediatric Society

President: Dr. Harry S. Altman, 940 Grand Concourse, New York.
Secretary: Dr. David Zahn, 2474 Valentine Ave., New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

Brooklyn Academy of Pediatrics

President: Dr. Benjamin Kramer, 6 Pierrepont St., Brooklyn.
Secretary: Dr. John A. Monfort, 861 Carroll St., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Fourth Wednesday of each month, except May, June, July, August and September.

Central New York Pediatric Club

President: Dr. Frank van der Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Third Tuesday of April and September.

Chicago Pediatric Society

President: Dr. Gerard N. Krost, 2376 E. 71st St., Chicago.
Secretary: Dr. George F. Munns, 723 Elm St., Winnetka, Ill.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

Dallas Pediatric Society

President: Dr. Edgar Loomis, 351 W. Jefferson Ave., Dallas, Texas.
Secretary-Treasurer: Dr. John E. Ashby, 3610 Fairmont Ave., Dallas, Texas.
Place: Bradford Baby Hospital. Time: 1 p. m., second and fourth Saturdays of each month.

Detroit Pediatric Society

President: Dr. William O'Donnell, 1553 Woodward Ave., Detroit.
Secretary: Dr. Robert Mason, 5140–2d Blvd., Detroit.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month from October to June, inclusive.

Fulton County Medical Society, Pediatric Section (Atlanta, Ga.)

Chairman: Dr. Stephen C. Redd, 157 Forrest Ave. N. E., Atlanta.
Secretary: Dr. C. Dixon Fowler, Medical Arts Bldg., Atlanta.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: Second Thursday of each month from October to April, 8 p. m.

Houston Pediatric Society

President: Dr. Thomas J. Donovan, 3820 Fannin St., Houston, Texas.
Secretary: Dr. Raymond Cohen, 2300 Caroline St., Houston, Texas.
Place: Houston Club. Time: Third Monday of each month.

Kansas City (Missouri) Pediatric Society

President: Dr. John Aull, 1103 Grand Ave., Kansas City.
Secretary: Dr. George Herrman, 1107 Bryant Bldg., Kansas City.
Place: Kansas City General Hospital. Time: On call.

Los Angeles County Medical Association, Pediatric Section

President: Dr. H. R. Cooder, 3875 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. Paul M. Hamilton, 248 E. Main St., Alhambra, Calif.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd.
Time: Second Monday of February, April, June, October and December.

Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Benjamin Stoloff, 1149 Eastern Parkway, Brooklyn.
Secretary: Dr. Harry Apfel, 988 Park Pl., Brooklyn, N. Y.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., second Monday of each month, October to March, inclusive.

Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. James Dobbins, 42-04 Ditmars Blvd., Long Island City, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144–38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry H. Donnally, 1612 I St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Harry A. Spigel, 2647 Connecticut Ave. N. W. Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of October, January, February, March and April.

Memphis Pediatric Society

President: Dr. Barton Etter, 376 S. Bellevue St., Memphis, Tenn.
Secretary-Treasurer: Dr. Harry Jacobson, 1193 Madison Ave., Memphis, Tenn.
Place: John Gaston Hospital. Time: Quarterly.

Milwaukee Pediatric Society

President: Dr. S. F. Morgan, 5630 N. Lake Dr., Milwaukee.
Secretary-Treasurer: Dr. F. R. Janney, 1411 Wauwatosa Ave., Wauwatosa, Wis.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Rustin McIntosh, Babies Hospital, New York.
Secretary: Dr. Leslie O. Ashton, 850 Park Ave., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Northwestern Pediatric Society

President: Dr. Roland E. Nutting, 205 W. 2d St., Duluth, Minn.
Secretary-Treasurer: Dr. John M. Adams, 1009 Nicollet Ave., Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

Oklahoma City Pediatric Society

President: Dr. A. L. Salomon, 1200 N. Walker St., Oklahoma City.
Secretary: Dr. G. R. Felts, 1200 N. Walker St., Oklahoma City.
Time: Every month, except June, July and August.

Philadelphia Pediatric Society

President: Dr. Pascal F. Lucchesi, 4000 N. Front St., Philadelphia.
Secretary: Dr. Aims C. McGuinness, Children's Hospital, 1740 Bainbridge St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to April, inclusive.

Pittsburgh Pediatric Society

President: Dr. E. L. Piper, 3200–5th Ave., Pittsburgh.
Secretary-Treasurer: Dr. Carl Ruder, 646 Washington Rd., Mount Lebanon, Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate month from October to June, inclusive.

Richmond Pediatric Society

President: Dr. Charles L. Outland, 312 N. 9th St., Richmond, Va.
Secretary-Treasurer: Dr. T. Stanley Meade, 913 Floyd Ave., Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: 8 p. m., third Thursday of each month, except June, July and August.

Rochester Pediatric Society

President: Dr. David Wolin, 16 N. Goodman St., Rochester, N. Y.
Secretary: Dr. W. L. Bradford, 16 N. Goodman St., Rochester, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Second Friday of each month.

St. Louis Pediatric Society

President: Dr. Max Deutch, 634 N. Grand Blvd., St. Louis.
Secretary-Treasurer: Dr. Peter G. Danis, 634 N. Grand Blvd., St. Louis.
Place: St. Louis Medical Society Bldg. Time: Third Friday of ech month from September to June.

Seattle Pediatric Society

President: Dr. Jay I. Durand, 1305–4th Ave., Seattle.
Secretary: Dr. Frederick B. Joy, 1215–4th Ave., Seattle.
Place: College Club. Time: Third Friday of each month, 6:30 p. m.

Southwestern Pediatric Society

President: Dr. Norman Nixon, 1136 W. 6th St., Los Angeles.
Secretary: Dr. Donald C. Shelby, 3780 Wilshire Blvd., Los Angeles.
Place: Jonathan Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Yark Osterlin, Central Michigan Children's Clinic, Traverse City, Mich.
Secretary: Dr. D. Murray Cowie, University of Michigan, Ann Arbor, Mich.
Time: Nov. 15-16, 1940.

Westchester County Medical Society, Pediatric Section (New York)

President: Dr. M. Bernard Brahdy, 531 E. Lincoln Ave., Mount Vernon.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 Highland Ave., Rye.
Place: Grasslands Hospital, Valhalla, N. Y. Time: Third Thursday in October. December, February and April.

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